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in round table on causes of mental disorders: a review of epidemiological knowledge, 1959

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IN THIS ISSUE

THE great and growing interest in mental disorders, especially in their prevention, has stimulated much epidemiological research designed to obtain evidence on the causes of different kinds of mental disorders which can be the basis for developing preventive programs. Aware of this interest and of the many recent studies which have evaluated data on associations between a variety of factors and mental disorder, in 1959 the Milbank Memorial Fund sponsored a Round Table meeting at Arden House, at which present knowledge about causation of mental disorder was discussed. In preparation for this meeting, eight distinguished authorities were asked to prepare review articles summarizing the evidence relating to different kinds of causes which had been thought to lead to mental disorders. The papers were distributed to the participants in advance of the meeting. At the meeting, the discussion of each review paper was opened by a previously designated participant; a general discussion followed; and the reviewer then added his own comments on the discussion.

The Fund's formal publication of the Proceedings began in the January, 1961 issue of the Quarterly (Vol. xxxix, No. 1), and is scheduled to be completed in the July issue. Included in these Proceedings are the review papers, the opening discussions, summaries of the subsequent general discussion, and the closing remarks of the authors of the review papers. The first paper of the meeting—"Genetical Etiology in Mental Illness" by Professor Jan A. Böök—was printed separately from the Proceedings series in the July, 1960, issue of the Quarterly (XXXVIII, No. 3), so that, unfortunately, the discussion of the paper was not published at that time. However, this material will be added to the Proceedings which will be collected in a

volume entitled: CAUSES OF MENTAL DISORDERS: A REVIEW OF EPIDEMIOLOGICAL KNOWLEDGE, 1959, sometime in the latter part of 1961.

The reader is referred to the January, 1961, issue of the *Quarterly* for a brief introductory statement outlining the objectives of the meeting and for the abstracts of all of the review articles presented at the meeting.

Three of the review papers, together with their discussion, will be found in this issue:

The Family and Mental Disorder:
An Incomplete Essay. John Cumming, M.D.

This "incomplete essay" (so-called since it reviews only a portion of the very large number of studies which have tried to link the development of a mental disorder with a particular characteristic of the patient's family or family member) goes into less detail in criticizing the methodology of these investigations than in pointing out that the questions have often been too imprecisely stated to make research productive of answers. Dr. Cumming's discussion seeks to mark out a path through the present chaos in the field and to suggest ways of relating the studies to one another so that future investigations will be more productive.

Proximal or Precipitating Factors in Mental Disorders:
Epidemiological Evidence. D. D. Reid, M.D.

This review concludes that the field lacks any sustained and systematic approach to the problem of precipitating factors. Dr. Reid believes that while the most valuable studies have been made of stress in wartime, events which occur in civilian life have not been absolved: they have only been insufficiently examined.

Social Structures and Mental Disorders:
Competing Hypotheses of Explanation. H. Warren Dunham

The evidence which links an individual's position in the social structure in which he lives to the risk of manifesting men-

tal disorders is examined; and a number of critical studies are woven into a reasoned discussion of both methodological and theoretical issues. From this evidence it is clear that the relationship between social class position, occupation, and a number of other similar factors, and the frequency of certain mental disorders (in one or another form of treatment) is very marked and well established. The causal meaning of these associations is still uncertain. The problems connected with exploiting the clues suggested by these associations, and the problems involved in closing the gaps in our knowledge about them, are amply classified and clarified, both by Dr. Dunham and by the group in the ensuing discussion.

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Higher death rates experienced by males than by females frequently have been attributed largely to biological factors. This excess mortality for males has been increasing at most adult ages in recent years in the United States when mortality for both sexes has been declining. In an article entitled "Causes of Death Responsible for Recent Increases in Sex Mortality Differentials in the United States," Philip E. Enterline describes trends in specific causes of death for different age groups and suggests that environmental factors may have an important role in the widening sex differential in mortality. The largest increases in the sex mortality ratios are found for age groups 15 to 24 years and 45 to 64 years. Increases in the sex mortality ratios are largely the result of recent trends in rates for a few important causes of death for which trends are of two types: "declines in causes of death importantly affecting females (tuberculosis, maternal mortality, cancer of the uterus and diseases associated with high blood pressure), and increases in male death rates for motor vehicle accidents, lung cancer, and coronary heart disease."

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A paper, "Implications of Prospective United States Population Growth in the 1960s" is contributed by Dr. Joseph S.

Davis. Among the prospective developments considered are increases in school enrollments, number of women of reproductive age, size of the labor force, and number of aged persons. He discusses the resurgence in fertility, especially among nonwhites. The important trends in population redistribution during 1950–1960 include the marked gains in California, Florida, Texas, New York, and Ohio, and the losses in Arkansas, Mississippi, and West Virginia. A leading implication, as seen by Dr. Davis, will be a marked increase in demand for consumption goods and services. The relatively large increase in nonwhites enhances both the necessity and the difficulty of removing the economic and social handicaps confronted by these groups. Among the other problems are the utilization of the increased labor force, the provision of housing, the burgeoning of suburbs, and the increased need for investment capital. The author states that "Americans are accustomed to rise to challenges, and . . . our economic and social history has typically confounded both superoptimists and pessimists of all degrees."

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In an article in the July 1955 issue of the *Quarterly* Dr. M. A. El-Badry presented some demographic measurements for Egypt based on assumptions of stable age distributions. It was stated that the technique might be applicable to other underdeveloped areas lacking vital statistics but having a history of approximately stable fertility and mortality and an absence of migration across national boundaries. In view of the recent declines in mortality in the face of persistence of high fertility in underdeveloped areas, Dr. S. H. Abdel-Aty has found it more realistic to assume "quasi-stable" rather then stable populations in such places. His results are presented in this issue in an article "Life Table Functions for Egypt Based on Model Life-Tables and Quasi-Stable Population Theory."

THE FAMILY AND MENTAL DISORDER: AN INCOMPLETE ESSAY

JOHN H. CUMMING, M.D.

THIS article does not pretend to summarize the vast amount of literature on the family and mental illness; and it completely disregards the largest single type of study: the family influences on the growing child as reported in case studies dealing with the psychodynamics of illness. Whatever their clinical value such studies contribute little to our scientific knowledge.

Because I believe that it is ineffectual to consider a subject apart from some theoretical approach to it, I have attempted to summarize a theoretical position about the family and to derive from this a series of categories of studies of the family.

Using the index case as a point of reference I have explored the different forms which the family takes as the index case moves through his life cycle. This approach is recommended by Lansing and Kish (30) who state "Advantages of family life cycle (as an independent variable) over age probably can be shown for many economic, social, political, and psychological variables. . . ."

I will set forth the theoretical functions of the family unit for its various members and try to discern which of these functions is being studied in various researches. Finally I will try to separate studies which deal mainly with the *function* of the family unit from those which deal with actual changes in its usual *structure*. The typology of family studies which these distinctions yield is heuristic because it not only divides up those studies currently in the literature but also points up those areas of research which are being neglected. Some of these areas seem to be promising for epidemiological research.

FAMILY THEORY AND CATEGORIES OF STUDIES

Most of the work which attempts to relate various aspects of

family structure and function to the occurrence of mental illness lacks any systematic definition of what is meant by a family. Researchers often use the word "family" in several different ways although this is seldom explicitly set forth.

Some refer to what is called the "family of orientation": the family unit into which the index member was born and which is responsible for his socialization. Others to the "family of procreation": the family which the index member forms by marriage and in which he will in turn raise his own children.

A third "family state" should also be distinguished because of its theoretical utility. This is the state of familial limbo or non-family which typically occurs between the time when the index member has broken most of his ties with his family of orientation and has not yet married and formed his own family of procreation. A similar state of familial limbo can be arrived at after the formation of the family of procreation through divorce, separation, or the death of a spouse.

As conceived here, families of orientation and procreation are all nuclear families, that is they contain at most two generations and have conjugal, parent-child and sibling relationships only within them. We will also, however, have to consider the extended family which is formed either by the addition of a third generation to the nuclear family group or by lateral extension, that is, the addition of more distantly related kin such as the uncles, aunts, grandparents, or cousins. In our society this probably occurs most commonly when a widowed parent goes to live with the family of procreation of his adult child. The implications of this type of family extension will be developed as we progress.

The American Family has undergone considerable change since the beginning of the present century. Many of the functions of the family have been taken over by other institutions in the society. This has been taken by some observers to mean that the family as an institution is disintegrating. Parsons (39), however, observes that not only is the family becoming a more popular state, that is, a greater percentage of people are mar-

ried at any given time, but it also retains two vitally important functions:

> We therefore suggest that the basic and irreducible functions are two: first, the primary socialization of children so that they can truly become members of the society into which they have been born; second, the stabilization of the adult personalities of the population of the society. . . . it is the control of the *residua* of the process of socialization which constitutes the primary focus of the problem of stabilization of the adult personality.

These two functions suggest further ways of categorizing studies of the family. Thus one category includes studies which focus on defects in the process of socialization. Such defects result in an imperfect internalization of the norms of society and a resulting tendency to deviant behavior. Another class of studies is implicitly or explicitly concerned with the stabilizing and controlling effect of family life on the adult individual and the causes of breakdown of this function. We might say, *vis-a-vis* mental illness, that the socialization studies concern themselves with the etiology of, or the formation of, a predisposition to mental illness while the studies of stabilization and control concern themselves with the precipitation of illness, with the process of hospitalization, and with readjustment subsequent to an episode of mental illness.

Parsonian theories of the family can be set forth in two ways: first, with an emphasis on the structure of the social group and second, with emphasis on its functioning. Structurally, it is obvious that the family is a small group and shares the general characteristics of such groups. The family, however, has certain structural qualities which set it apart from other small groups. Part of the difference stems from its function of socialization. This function, by definition, means that the family must teach its younger members not only the values of its own sub-system but also the values of the larger society. It must also, like other socializing institutions, be resigned to expelling its members when their socialization reaches an adequate level.

For the family best to accomplish its socializing function it is necessary to have the larger society "represented" in family interaction. The older members of the family, particularly the father, have roles in the larger society and in the ideal state they should represent these societal roles in the family setting. If the father does not adequately represent his occupational role in the family, the family will probably be insufficiently connected with the larger group and will tend to take on the qualities of a self-contained system, rather than retaining the characteristics of a sub-system of a larger organization. Such a change toward system functioning will have at least two consequences. First, the family will tend, like all system groups, to retain its members and try and gain primacy for its values over the more general values of society. Secondly, because of its exclusive nature, this family will discourage peer group relations for its children and will leave them inadequately prepared to form new group relationships outside of the family.

A situation where the father did not play such a representative role might occur if he were inadequate in his occupational role, or in some way clearly deviant and rejected by his associates in the larger society, or clearly dominated by an instrumentally oriented wife within the family. The consequences of such a failure on the part of the father or the family unit—that is in the family unit's subsequent retention of children and its exclusive attitudes—will be seen to have special importance when we consider power relationships within the family.

Such considerations are important in thinking about work such as that of Lidz (31, 32), since they explain why the affected member may have been held so long in a deviant family group. It might also allow us to generate some hypotheses to explain why some sorts of deviance on the part of parents seem to be damaging to children while others are not.

While the family is a sub-system, it is as Henry (26) pointed out in an early work, a system of interrelated groups in itself. The groups might be two-person groups such as husband-wife,

mother-child, sib-sib as well as three and four-person groups depending on size of the family. The number of possible groups goes up rapidly with increasing family size. Family size may indeed be an important theoretical variable in socialization studies because of the difference in the number of differentiated experiences which can be found within the confines of the family. Of the sub-systems of the family only the mother-child constellation has been subjected to any considerable amount of research. Recently, however, Henry, Spiegel (43, 44), Bowen (8) and Lidz, to mention only a few, have attempted either to see the family as an operating whole or else to view it as a system of sub-systems each of which merits attention.

The various sub-groups which form within a family differ from one another, however. This is because the family is by its nature a differentiated system, that is, the people in it are not only of different ages and sexes but have different, though complementary, roles. All families are divided along two great axes. Because of the generational differences which exist in any family and because of the prolonged state of helplessness of the human infant, it is obvious that the father and mother can be designated as having more power than the children. A second axis of role differentiation corresponds with sex categories. The male members play predominantly instrumental roles, that is, they provide for the families' needs in a practical way and relate the family to the surrounding community. They represent the family as it strives to achieve its goals, and defend it from any threat from outside. The women of the family play predominantly socio-emotional roles and are primarily responsible for dealing with the tensions which arise within the family, and thus with defending the system against internal threats to its integrity. These two axes, taken together, yield four descriptions of the roles within the family. They give us the dominant role characteristics of the father, instrumental and powerful; the mother, powerful and socio-emotional; the son, less powerful and instrumental; and the daughter, less powerful and socio-emotional. According to this scheme, important disruptions in

family functioning would occur if one or another member did not fulfill his or her appropriate function, or if competition for roles arose between family members.

Such are some of the very general structural considerations which seem to be of importance for the functioning of the family as we know it. There are other ways, of course, of viewing the functioning of the family in its socialization task. Perhaps the best known and most used is the Freudian concept of stages of development, both in its original form and as modified by Erikson (15). A more interactive sociological model can be derived from Sullivan (50) or George Herbert Mead (35). Piaget (40) furnishes us with brilliant empirical descriptions of the development of the thinking of young children. Parsons, in the work which we have been reviewing, presents a sociological equivalent of the Freudian phases in which he sees the stages of development as a series of splits in the types of roles which the child experiences and internalizes. In the earliest phase of development, during "oral dependency," he sees the mother and child as an identity. As this phase moves into anality, the mother and child begin to interact as separate individuals and by the time of the Oedipal phase these two roles have divided into the four whose structural aspects were described above. The next discrimination made by the child is between roles which are typical of the family and therefore are *particularistic,* in Parsons' terms, and those characteristic of the wider non-familial world which are governed by *universalistic* criteria. Finally, in the phase of adolescence, the individual learns to distinguish between those roles which demand from the role holder inherent attributes, or *quality,* and those roles characterized by what the role holder has accomplished or by his *performance.* Parsons claims that at his high level of generalization the ability to act in, and distinguish among, these differentiated roles constitutes adulthood. The Parsonian view of the functioning of the family is neither particularly easy to grasp, nor, to those schooled in the more colorful psychoanalytic theories, very appealing. It does have, however, the great advantage of being conceptually

related to his idea of family structure. As we review the extant research into the connection between the family and the occurrence of mental illness, we will come to see how important this sort of relationship is. For the moment, however, it is sufficient to realize that it will be useful further to attempt to classify researches into those concerned primarily with the functioning of the family, and those concerned primarily with its structure.

We will now consider some of the studies in the family. To some extent we will use the theory we have just outlined as a base line for our criticisms and for suggesting areas for further research. Further, the conceptual scheme which we have outlined allows us to make a categorization of studies which we hope will be heuristic. By naming the types of family situations which an index case may experience as he passes through his life cycle and dividing each one of these phases, first, by the primacy of the family function which is being studied and, second, by the approach that is being used, we would arrive at a series of categories as shown in Table 1. It should be noted that this categorization is not exhaustive, for instance the difference in the nuclear family condition as opposed to the extended family condition is considered only for the family of procreation. Similarly, separate sections in the text are set up for each cell only for the family of orientation, since the quantity of research in some of these areas has been small. I have tried throughout, however, to organize the paper according to these principles.

Table 1.

	Socialization		Stabilization-Control	
	Function	Structure	Function	Structure
Family of Orientation	Section I	Section II	Section III	
Familial Limbo-State	Section IV			
Family of Procreation	Section V			
Familial Limbo-State	Considered with Section IV			
Extended Family of Procreation	Section VI			

Section I

FAMILY OF ORIENTATION. SOCIALIZATION: FUNCTION

The overwhelming majority of all studies of the family have used the child or young adult as the index case. They consider the child in relation to his parents and are concerned with the formation of the adult personality rather than with stabilization-control or tension management. Further, they are studies of the process involved rather than studies of the effects which changes in the form of the family might have on these processes.

Unfortunately, almost all of these studies are unsatisfactory. Spiegel and Bell (45) in reviewing 85 such studies point out that only 17 of the total group employ control groups. Seventy of their group of studies (they selected only the largest of the hundreds available) were "clinical" rather than "objective." The "objective" studies, which were also by-and-large the group who used controls, have tended to contradict the findings of the clinical studies. Stevenson (49), in a paper in which he examines the assumption implicit in many of these studies—namely that the child is more plastic and subject to influence than the adult—points up the studies by Orlansky (38), Thurston and Mussen (52), and Sewell (42) which indicate that we cannot accept the idea that child training and early life experience have specific effects on adult personality. Thorne (51), on the other hand, by following the genealogy of two families through four generations, makes an excellent case for the thesis that some behavior patterns are learned, in this case patterns of hostile aggressive behavior.

Many of these studies are investigations into the family background of schizophrenics. The index case in these studies is often a young male schizophrenic and the focus has been the relationship of this person with his living parents. Usually the patient has been living at home at the time of hospitalization. As a matter of fact we suspect that the young male schizophrenic living in his family of orientation at the time of admission to hospital is not the modal case. While this does not pre-

vent these studies from contributing to knowledge about the etiology of schizophrenia, it does diminish their usefulness in determining the precipitating factor in the acute illness or the forces involved in hospitalization. Lest I be accused of setting up a straw man, let me hasten to say that almost all of these studies do focus on etiology. Since functional studies are difficult and time consuming, it is almost impossible to study a group large enough for adequate statistical analysis. The two largest of these studies, those of Lidz and Spiegel, have study groups of fifty and eighteen cases respectively. Bowen, Bateson (5) and Wynne (55) have made studies which all fall short of these numbers. Spiegel's research is the only one in which controls are used. However, while the remainder of the researches are concerned with schizophrenia, Spiegel deals with children at a child guidance clinic, suffering mainly from neurotic disorders. Thus we have no controlled functional studies of schizophrenia and our judgment of the usefulness of all of these family studies is limited by the fact that we can only compare the families of schizophrenics with those of our own and those of our friends.

The cheering (and from the point of view of the statistician, the dubious) feature of these studies is their claimed ability to turn up universals. Bowen, for instance, finds what he terms "emotional divorce" uniformly present in the families of his schizophrenics. The father and mother maintain a "striking emotional distance" from each other. Coupled with this, he notes an inadequate-overadequate reciprocity which arises from immaturity on both their parts. This pattern is also observed by Vogel working on Spiegel's families of neurotic children. He has called the process "polarization" and believes that the parents have similar neurotic conflicts marked by ambivalence, and in their interaction each tends to support one side of the ambivalence. A move of either from his position will bring about a shift in the other to restore balance. In Bowen's, Wynne's and Spiegel's cases, the families are often described as having a superficial and spurious agreement about their problem areas.

The similarity of Spiegel's findings with those of the remainder of this group is somewhat distressing. If the family backgrounds of neurotic children and young schizophrenics are so similar then we may have to assume that such conditions are necessary but not sufficient causes. Roberts and Myers (41) contribute to a solution of this difficulty by comparing the background of two groups of schizophrenics and neurotics. The groups were different in their classes of origin but each contained equal numbers of the two diagnostic categories. Their investigation allows them to specify not only the kind of stress but the quantity of such stress in the background of each case. They claim that while the backgrounds of their schizophrenic and neurotic cases were similar, the schizophrenics had consistently a larger amount of stress in their background. Throughout this book schizophrenia is treated as if it were a bad case of neurosis. While this interpretation may be consistent with the regression theory of schizophrenia, it is inconsistent with the more recent theories of this illness stemming from the ego-psychology of Federn (17) and others.

Roberts and Myers' book is disappointing to those of us who eagerly awaited its printing. The authors use very little general theory and for the most part are content to use psychoanalytic constructs to explain their findings. Their data are not presented in a form which makes it possible for the reader to rework them if he wishes to do so. Finally, there seem to be methodological errors. An interesting portion of the book rests on a comparison of the social mobility patterns of two groups of patients from Social Class III and Social Class V respectively. Many of the Class III patients had parents who were in a lower social class, Class IV. On the other hand, Class V patients who come from the lowest social class could not by definition have parents who were lower in their social position than the patients. It would seem probable that the psychological impact of mobility patterns would differ between these two groups not only because of their class backgrounds and their illnesses but also because of their family mobility histories. In this latter im-

portant area the two groups do not seem to be comparable.

Lidz's work deserves special mention because of its monumental proportions. It is a great pity that he did not control his large sample. His results, however, are so striking that they command attention. He claims that in every case of schizophrenia studied there was one parent who was an unhospitalized psychotic or very seriously neurotic. It is his thesis that the psychotic symptomatology is learned from this parent. Unfortunately, we cannot be sure that this result does not come from his sample selection.

All the authors which we have mentioned have offered plausible formulations of the familiar origins of mental illness. Further, all of these studies have similarities and their findings tend to support one another. None of them would satisfy an epidemiologist, however.

Section II

FAMILY OF ORIENTATION. SOCIALIZATION: STRUCTURE

Work on maternal deprivation[1] seems to fall naturally into this class of studies. When the structure of the family has been disturbed in such a way as to make the mother unavailable to the child, dramatic signs of pathology are said to ensue. Bowlby (9), Spitz (47, 48) and Bender (7) have all written extensively about maternal deprivation, with Bowlby's work remaining the most comprehensive. There is an assumption in most of these cases, probably correct, that the etiologic trauma is the separation of mother and child and not some other stress in the total situation. It is this assumption which has allowed various workers to group studies of hospitalization of children, death of the

[1] Since the writing of this section two important references have come to my attention. Barbara Wootton in her excellent book, Social Science and Social Pathology, MacMillan, New York, 1959, has a chapter entitled "Theories of the Effects of Maternal Separation or Deprivation." This critical analysis of the literature on Maternal Deprivation presents my point of view better than I can hope to. Secondly, the work of Harriet Rheingold as published in Monographs of the Society for Research in Child Development Inc., Vol. XXI, No. 2, 1956, *Child Development* 1959, 30, and the *Journal of Comparative and Physiological Psychology*, Vol. 52, No. 1, 1959, seems to offer a fresh new approach to this problem, an approach which has a chance of producing a real advance in knowledge.

mother, and effects of mother working, under a single rubric. From our point of view, it seems better to consider those cases in which the child is removed early from the entire family (an early case of familial limbo) separately from those cases in which the child has been left within the family and the family disrupted by the death or desertion of the mother. From a structural point of view, while it seems probable that a substitute mother role might be provided in the family setting, it would be difficult to provide it in a situation such as an orphanage, where the father role—a necessary support to the mother role—is lacking.

I will not even attempt to summarize the findings of these studies. I will, however, quote from a recent as yet unpublished manuscript[2] to illustrate the magnitude of the claims which are being made.

> When the symbiotic relationship between a child and his mother from six months to three years is disrupted for more than a day, typical physical symptoms develop: insomnia, lack of appetite, weight loss, predilection to intercurrent infections, retardation of physical growth, backwardness in talking, restricted activities with the child sitting and lying inert in a dazed stupor. Emotionally, the child is depressed, apprehensive, sad, and withdrawn. He rejects the environment and makes no attempt to contact strangers. He is at first acutely and inconsolably distressed for days, a week or longer without a break. He shows agitated despair with screaming and moaning. He refuses food and comfort. Only exhaustion brings sleep. After days, he becomes quieter and may lapse into apathy. He may regress to infantile modes of behavior, i.e., wetting, soiling, masturbation, refusal to talk and to walk. Intellectually, the longer the child is separated, the lower his developmental Quotient falls. This seems to be a consistent finding in all countries. Socially, his limpet-like attachment to his mother is such that only if she is with him or nearby can he manage his environment and himself. All effort at "separation in slow stages" is in vain. How is

[2] Prepared by Dr. Mary Mercer for the Subcommittee on Primary Prevention Committee on Mental Health, Technical Development Board, American Public Health Association.

it possible to reconcile him to the loss of that vital part of himself, his mother?

These statements are made by-and-large on the basis of the group of studies which have been so well summarized by Bowlby. These studies command attention not so much because of their excellence in design (on the contrary, many of them have obvious flaws) but from the consistency of direction of their results.

In distinction to the studies which claim massive and irreparable damage resulting from loss of the mother, one has to contrast the extreme cases described by Kingsley Davis (13) in which recovery was remarkably quick and complete after a long period of extreme isolation. We should also consider the results of different methods of child raising.

Spiro (46), in a study of one Israeli kibbutz, reports that the education and socialization of the kibbutz children is the function of their nurses and teachers, and not of their parents. Infants are placed in infants' homes upon the mothers' return from hospital where they remain in the care of nurses. Both parents see the infant in the nursery, the mother when she feeds the infant, the father upon his return from work. The infant is not taken to its parents' room until its sixth month, after which it remains with them for an hour. As the child grows older, the amount of time he spends with parents increases and he may go to their room whenever he chooses. Usually the amount of time spent with them is a two hour period at the end of each day and a longer period on the Sabbath.

It seems to be safer at present to assume that what these studies indicate is that children who are raised in institutions develop differently from those who are raised in nuclear families. The precise nature of the difference can be accounted for by the nature of the individual institution.

It may not be out of place here to enter a plea for a more precise definition of terms in these studies. Ainsworth and Bowlby have attacked this problem in their excellent monograph, "Research Strategy in the Study of Mother-Child Sepa-

ration" (3). However, there still is in many studies a confusion between the results of actual enforced physical separation, with its consequent cessation of interaction, and temperamental or emotional distance between people. Thus Kohn and Clausen (27) in speaking of isolation and schizophrenia treat isolation as if it were lack of sociability. They find that this variable is no more strongly related to schizophrenia than it is to manic-depressive psychosis. In a further study of shyness and withdrawal, Michael (36) in an impressive long-term follow-up study of children who were seen at a child guidance clinic points out that introverts have a lower than expected incidence of schizophrenia in adult life. In studying the family, however, we should be careful that we do not confuse conceptual entities, such as Bowen's "emotional divorce" which is a description of impaired family function, with actual family disruption which is a structural change.

The more obvious aspects of family structure as it relates to mental illness, such as the ordinal position of the child in the family, or family size, have been unpopular as research problems in recent years. Malzberg (33) apparently convinced us that ordinal position did not influence the rates of hospitalization. Myers and Roberts have recently challenged this view, but I am not convinced that their methods are as useful as that of Malzberg in his earlier studies. Wahl (53), in a study of the background of 583 cases in the United States Navy, reported that schizophrenics came much more often from large families. However, it seems obvious that he had neglected to consider that large families present a larger group to the risk of schizophrenia. When corrected for family size, his differences seem to disappear.

Wahl also presented evidence that in his sample there was a high incidence of the loss of a parent by death before the patient attained his fifteenth birthday. He reviews a number of other papers which, in the main, support these findings. On the other hand, it is obvious that only a small number of persons who suffer the loss of a parent are hospitalized for mental illness. It

would be of great interest to know, in structural terms, if the schizophrenics in Wahl's sample came from homes where the role of the missing parent had not been filled by some other person. Gerard and Siegel present data which might be construed as casting doubt on Wahl's findings. However, since they had a high loss from their sample because of inability to locate reliable informants—in most cases fathers and mothers of the patients—one may legitimately suspect that broken homes were under-represented in their sample.

The Gerard and Siegel (21) study almost bridges the category of structural and functional studies. Their variables might be referred to as "family traits." They find their sample of urban male schizophrenics characterized by a markedly heightened relationship with mothers. The mother was usually a clearly dominant person in the household and the fathers were disinterested or absent. This mother dominance distinguished these families from a group of controls. The mothers of the schizophrenic males had markedly overprotective attitudes and the schizophrenics were more often spoiled and pampered than were the controls. Many more schizophrenics than controls lived in neighborhoods in which their families were markedly different either ethnically, economically, or religiously from their neighbors. However, differences in the toilet training and breast feeding of the schizophrenics were in the direction opposite to that usually postulated. The schizophrenics were breast fed longer than the controls and the toilet training of the two groups did not appear to differ. The authors used as controls the members of a graduating class in a local high school. Serious objections can be raised to this control group. Despite this, the study is one of the more adequate ones in this field and deserves attention.

Kohn and Clausen (28) whose work merits attention by virtue of its close attention to the requirements of method have pointed out that parental authority behavior varies more between social classes than it does between the parents of schizophrenic patients and others.

Section III

FAMILY OF ORIENTATION. STABILIZATION-CONTROL: STRUCTURAL AND FUNCTIONAL STUDIES

Structural and functional studies of the stabilization and control aspects of the family of orientation are almost non-existent. Some aspects of general studies of child raising touch on these points but without any conceptual separation of them from the problems of socialization. This is undoubtedly a reflection of our current interest in child raising and of our child-centered culture. It is inconceivable that neglect of this area of research could have taken place fifty years ago when a different view of children existed. One crucial area for study suggests itself. If the family of orientation is unduly[3] prolonged in time, certain problems inherent in family structure and in our society manifest themselves, particularly in relation to the male members. We have already pointed out that there is an important power differential between the generations of the nuclear family. In the father's case this power is derived by-and-large from the prestige attached to the occupational role which he plays in the community and which he represents within the family. In our North American society there is a strong pressure for young men to demonstrate that they can get and hold jobs. This is a value even in those situations where it is not economically necessary for them to work. On the other hand, once the male child takes a job he, as well as his father, represent occupational roles within the family. Consequently the power gap between father and son is abruptly narrowed. Such a shift in relationship often results in stress and dissension. The stabilization and control function of the family for the son will automatically be lessened.

In discussion of theory I pointed out that the family must remain a sub-system of the total society. If it takes on too many of the aspects of a self-contained system, it tends to retain its

[3] Objection has been raised to the word "unduly." We use it to imply that in this culture there is probably an optimal amount of time which should be spent in the family of orientation.

members unduly long and to prepare them inadequately for membership in alternative groups. Further, I pointed out that families are likely to become self-contained systems when the oldest male member—who is the main connecting link with the community—plays his representative role inadequately. When there is such an instrumentally inadequate father there is an enhanced chance of conflict when the son takes a job and becomes fully instrumental. Thus the male child as he matures in such a setting is in a true dilemma. If he stays home, the family fails him in its stabilization and control functions. If he leaves home, his social inadequacy makes it harder for him to form new group relationships thereby replacing the family with a functional equivalent. He is likely to remain an isolate and thus still be in a position of inadequate stabilization and control. These speculations lead us directly into a discussion of the Familial Limbo-State.

Section IV

THE FAMILIAL LIMBO-STATE

While this state is not often thought of as a variant of the family, some researches bear upon it. Malzberg (34) laid a firm basis for these in his studies of married and single states related to the incidence of various mental illness (hospital admissions). He says, "The evidence seems clear that the married population had, in general, much lower rates of mental disease than any of the other marital groups." Like several of the predecessors whom he quotes, he is inclined to attribute the high rates of mental illness among single people to a sort of low vitality which is at once a cause for the failure to marry and for the predisposition toward mental illness. He admits, however, that another explanation for the high rate of illness among the widowed is needed, since it cannot be assumed that they too lack "mental vigor." While not disputing Malzberg's findings, it seems that we should be able to find hypotheses both more theoretically sophisticated and more empirically testable than the above.

Adler (2), using another population, addressed herself to the question of whether marriage protects against illness or whether the prodromal signs of illness prevent marriage. Computing rates for various marital status groups at the estimated time of onset of illness, she concludes that the married have lower rates of psychosis even when computed on the basis of the marital status before the onset of the mental illness. However there did seem to be evidence that among the single some had not married owing to the prodromal symptoms of mental illness.

Adler further reports that while marriage does not appear to result in a higher recovery *rate*, her evidence shows that at follow-up the married had higher recovery level scores than the single. She points out, however, that this may have been caused by the marriage of certain patients after they left hospital. The living arrangements of the single patients are not specified.

Some of the studies of isolation are relevant to our consideration of the non-familial limbo-states. Faris first suggested that isolation had a causal connection with mental illness. He and Dunham in their studies of the ecology of the hospitalized mentally ill from Chicago (16) found especially high rates in the so-called zone of transition, the area of cheap hotels and rooming houses close to the center of the City. They felt that the high degree of isolation which they assumed to be associated with living in such areas was the causal factor most closely associated with the high rates of illness, particularly schizophrenia. Gerard and Houston (20), in an investigation of 305 male first admissions from the city of Worcester, Massachusetts, confirmed that there was a high negative correlation between a "favorability of living index" of an area and rates of first admissions for schizophrenia, but found that this difference was caused by the relatively large numbers of single, separated, and divorced men living alone in these unfavorable areas. Since these solitary males showed high geographical mobility it was assumed that they had drifted down into the zone of transition. However, since Gerard and Houston did not have a residential history for this particular group, there was no evidence that

either these men had moved down from better areas rather than simply moving frequently within the area and, further, that the movement was greater than that among solitary men unaffected with schizophrenia. The "drift hypothesis" was questioned further by Roberts and Myers (41) startling finding that the majority of schizophrenics are upwardly mobile from their families of orientation—in fact more upwardly mobile than their siblings.

Hare (24, 25) attempted to replicate Gerard's study on a large English population, the city of Bristol. In his work it is clear that, of the various criteria which he used to rank his city areas, the percentage of single-unit dwellings gives the best prediction of the rate of schizophrenia. Rates were high in both central slum areas and central middle-class areas. What these areas had in common was a large number of people living alone. Furthermore, the admission rates for schizophrenics who were living away from their families were highest from these areas. Unfortunately, the fact that a person is living away from his family does not necessarily mean that he was living alone. Hare's studies demonstrate a great need for determining rates specific to the populations at risk in various kinds of living situations. Not only must living alone and living with others (non-family) be analyzed separately, but the parental and conjugal living situation need to be dealt with separately.

Hare also investigated a small group of people living away from their families and concluded that about half of them—they seem from their description to be paranoid—had left the family because they could not get along in it. This did not mean that they were inadequate or downwardly mobile. They moved into central districts because that was the district of single-unit housing. While Hare feels that poverty is not a particularly strong factor, it seems to me that an estimate of the population at risk made from his figures suggests that among that group who live away from their families there is a much higher incidence in the poorer areas than in the middle-class areas. Both rates, however, are higher than those who live in a family set-

ting. Hardt (23) in a careful investigation of a New York State population confirmed Hare's general thesis and further demonstrated that the differences are not due to the age distribution of the populations within particular census tracts. This is an important variable to control in such studies.

Section V

THE FAMILY OF PROCREATION. SOME ASPECTS OF STABILIZATION, SOCIALIZATION, STRUCTURE AND FUNCTION

So little has the family of procreation been studied that we will consider all of these categories together. Several authors (Erikson (15), Parsons, Spiegel and especially Naegele (37)) have pointed out that the raising of children has a socializing effect upon the parents. It is popularly recognized that there are stages in the family of procreation: there are "newly-weds," "the young family" and finally, "the empty nest." Divorce rates are known to be related to the duration of marriage, reaching their highest point in the third year of marriage (54) and divorce diminishes in frequency with the number of children (4). However, I know of no one who has attempted to study duration of marriage and number of children in relation to the incidence of mental illness. While the loss of a parent is supposed to be important in the psychic economy of a child, and the consequences of the lack of a same-sex role model is often commented upon, there exist few studies of the change of role which occurs in one parent when the other one dies. That the effective instrumentality of the adult male role depends on the socio-emotional stability provided by the female role is assumed, and the reverse is to some extent recognized, but any precise study of this reciprocity of roles is lacking. I expect that when someone does study the role of the surviving spouse, it will be in the interests of determining the effect of these changes on the child. Such is the strength of the implicit belief that nothing very important happens in socialization after the seventh year.

As Parsons points out, the socio-emotional outlet provided by children has a stabilizing effect on the adult members of the

family. Again, virtually no studies have been made of the mechanisms whereby the stabilizing effects of family life are maximized and what sorts of circumstances can interfere with this function. Bowen's concept of emotional divorce is perhaps relevant, but the naming of a phenomenon does not explain either its genesis or its operation. What is the difference, for example, between a family which is filled with strife but can exist for years without emotional divorce and one in which strife results in this phenomenon?

There are two very important series of studies which deal with the post-hospital adjustment of the mentally ill and where the result of living with the family of procreation is compared with other living arrangements. The first of these is reported in a series of papers by Freeman and Simmons, the second is the work of Carstairs and Brown.

Freeman and Simmons (18, 19) started their investigation of a cohort of male functional psychotics discharged to the Boston area with the hypothesis that the paternal family would be more tolerant of deviance than the conjugal family. Thus, they expected that they would find (since a period of time had elapsed between the discharge of their cohort and the evaluation) that there would be more patients with a poor social and occupational adjustment living with their families of orientation than with their families of procreation. This, indeed, turned out to be the fact. They also allude to the fact that more patients had been returned to hospital from their conjugal families but they do not specify if this plurality is sufficient to account for the difference. Rate of return to hospital for those who went to parental and those who went to conjugal families was roughly equal (12). They further illustrated that patients seem to have a low level of adjustment when the female members of the household are rigid and punitive. In their families of orientation, the former patient is found to be socially and occupationally inadequate only when other adequate members were found in the household. Thus, while Freeman and Simmons demonstrate the greater tolerance of the family of orientation, there

are some indications that the behavior which the family is called upon to tolerate may be an acceptance by the former mentally ill person of the only role available to him. This possibility is considered by the authors (12) but evidently finds less favor with them than their tolerance hypothesis.

Brown (10) in a meticulous study reports similar findings but is able to extend them to demonstrate the active rather than the passive significance of the environment. He studied a group of chronic male schizophrenic patients (the criterion of chronicity being two years in hospital before discharge) and found that those who lived with siblings or in lodging houses after discharge succeeded much more often in remaining out of hospital than those who lived in hostels or in their families of orientation and procreation. In his findings one can see the same trends as Simmons and Freeman's report. Brown also found that more schizophrenics fail when returned to their wives than when they are returned from hospital to their mothers. But he finds that both of these environments are inferior therapeutically to the relatively neutral environments of living with siblings and living in boarding houses. Brown searched for factors present in the patients at the time of discharge but was not able to find any to account for the trend. He supports the often voiced opinion that families of orientation take patients from hospital who are not as fully recovered as those who are discharged to other settings but he is able to control this variable in his further analysis. Finally, he produces a number of very telling findings. Not only do sons in the family of orientation remain better integrated if they work, but they remain relatively better integrated if their mothers work and they do not. It seems that the symptomatology of sons who are returned to their families of orientation increases with the amount of time that they have to spend with their parents. There are findings which would indicate that wives are not very healthy for husbands either. One might wonder why there is such a marked difference reported between two familially-neutral settings, the hostel and the boarding house. There is a clue

in the author's description of the poverty stricken surroundings of the hostel. Despite my insistence that isolation is bad, and I have certainly emphasized this normative approach, it has been demonstrated in the Midtown (29) study that certain *wealthy* isolates are as healthy as the remainder of the top third of the economic group. To replace one normative idea with another (if one has to be isolated): it helps to be comfortably off financially. Both Carstairs' and Hare's work could be interpreted as supporting this idea.

Section VI

THE EXTENDED FAMILY

The effect of having an extended *versus* a nuclear family could be explored in relationship to any of the family types. I am, however, using it only to describe the case where the index person has been separated from his own family of procreation and enters another family of procreation, usually that of one of his children. The typical case of this process in our society concerns the older person. His own children are grown up and have left home. Then his spouse dies. At this point it seems typical for him to live alone for a period of time but in the end it is not unusual for him to take up residence with the family of procreation of one of his adult children.

The great contrast between this situation and that in which an adult child remains in the parental home lies in the different power positions in the two cases. When the aging adult joins his child's family, the child is the chief breadwinner or, if a woman, has the main socio-emotional task while the parent has to play a secondary role. This situation is dramatically different from what it was formerly. As before, the greatest conflict may be expected between father and son, when both are working, and the next greatest between mother and son. Daughters, with their greater role flexibility, may be better able to withstand the difficulties of this situation. This may be why, both normatively and actually, it is considered best for

parents to live with their female children if they cannot live alone.

Gruenberg (22) has pointed out that among elderly people the rate of hospitalization for mental illnesses of the senium was greatest in areas with a high percentage of the population living alone, even though, as the author points out, whether or not the elderly psychotic actually lives alone is still in question. Hare does not find this relationship between housing and hospitalization with diseases of the senium in Bristol, and believes a possible reason for the difference is the social legislation which provides so much assistance to the elderly in the British welfare state.

However, since Hare's study compares those living with their families with those not living with their families, his study is not strictly comparable to Gruenberg's. And neither study, because of the nature of the data, permits one adequately to examine the concept of isolation. Bellin and Hardt (6) in a study of symptomatology of mental illness in an elderly population found, however, that there were no differences in the amount of illness found among a group who were living with their spouses and those who had been widowed. Again for the widowed we lack information on their actual living status. As I suggested when I discussed schizophrenia, there is a need for a crucial study in this area focused on the interrelationship of the variables of hospitalization, amount of illness, family state, isolation, and economic condition, so that we can begin to understand the interpenetration of these variables.

The Family Unit

Several writers now support the view that it is impossible to consider any one person in isolation from his interrelated family network. This concept underlies Ackerman's (1) emphasis on "family diagnosis." Jules Henry has, from the viewpoint of the social scientist, demonstrated that the reactions of one member of the family can be understood only when viewed as the result of the effects of all the operant familial relations. As the num-

ber of persons in the family increases these relationships multiply very rapidly. Spiegel, who advocates a transactional approach, not only takes account of the number of relationships, but also shifts his focus of observation back and forth between family relationships, cultural values, and psychodynamics. This approach is so complex that he is often forced to use illustration instead of analysis. Bowen and Bateson seem to have decided (while they admit the importance of the family as a whole) that it is most important to focus on the parent-child triads. They believe this is the focus of most of the pathological interaction in the family.

I have by no means exhausted the subject of the family and mental illness here. I have not considered, for example, the family's reaction to the presence of defective children, and the psychiatric treatment of families or parts of families is beyond my scope. The research into the impact of mental illness upon the family is so well summarized by Clausen and Yarrow (11) that I will not resummarize it here. Mention should also be made of the careful study of Downes and Simon (14) who studied the characteristics of families of psychoneurotic patients. The striking finding of this study is the high secondary rates of psychoses and mental deficiency and even of chronic physical illness. There is a need for further work stemming from this excellent study. Some other aspects of the subject can be found in the chapter, "The Family and the Psychiatric Patient" by Spiegel and Bell in the recently published AMERICAN HANDBOOK OF PSYCHIATRY. Their formulations and their extensive bibliography should be as useful to others as they have been to us.

SUMMARY

It is clear that organized study of the area of the family and mental illness is in a state of chaos. I have attempted to categorize studies and to suggest ways in which the more important researches are related to one another. This process has forced attention to areas where there are logical research needs and suggested ways in which these projects might be formulated.

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Discussion

Dr. John H. Cumming: I would like to add a brief explanatory note on the "incompleteness" which is in the title. This incompleteness was quite deliberate. I didn't try to make a comprehensive coverage of the literature, first, because I was aiming at trying to

develop some sort of a conceptual scheme. A few studies which I thought were important I took to see how they fitted into the scheme and what additional ideas this process might generate.

The second point in the incompleteness is that the literature on the family would, I think, drown one if one tried to follow it through all the places where it could be found: in psychiatry, in the social sciences, in eugenics literature, in the literature on problems such as divorce, and in much educational literature. It is found in all those areas.

The final bit of incompleteness, and one that I hope we will get back to in this conference, is looking at the other side of the picture, which Dr. Densen brought up this morning.

While I have tried to introduce a systematic way of looking at the family, I think we still need to have a systematic way of looking at some of the diseases that we are trying to relate to the family, and Dr. Densen's comments on the reliability of diagnoses and such matters I think should come up for discussion.

I asked Dr. Carstairs, in our early correspondence, if he would enrich this with some of the British literature, and I think he is going to do so.

Dr. Morris Carstairs: I think we are all grateful to Dr. Cumming for having produced this chart and compass to guide us through the extremely widespread territory he has been assigned to cover in the literature.

I am sure we share with him a sense of inability to encompass all the different studies which have a bearing on the family, particularly since there is such a wide range of methods of approach and also of scientific quality.

I would like to recall the structural principles introduced near the beginning of his paper in order to place some additional studies in the chart that he has drawn up.

First of all, he lists different ways of looking at the family. There is the family of orientation into which one is born; then this idea of family limbo, when one leaves the parental family and hasn't set up a family of one's own; there is a group of studies concerned with the family of procreation; and finally, he draws our attention to the second limbo that can intervene when one's procreative life is over, when one is entering the later stages of one's career.

In his text he also reminds us that this isn't the sum of ways of looking at the family. There is also the family considered as the extended network of kinship. This has often been neglected in studies of our society, although it is a matter of great interest to anthropologists, and we are indebted to such people as Michael Young for reminding us that the extended family is by no means vestigial in contemporary urban communities. It still has quite a lively function, as Michael Young has demonstrated in Bethnal Green in the East End of London (24).

Young has recently informed me that while he was in California, early in 1959, he carried out a pilot study among middle class, fairly well-off households in Palo Alto. He found to his great surprise that contact with the older generation was still very much alive there, occurring with a regularity and frequency which was very far from what he had been led to expect.

Michael Young's collaborators have shown in subsequent studies that the extended family still has a significant function in times of crisis such as bereavement (15) and in old age (22). The latter study documented in detail the observation made in an earlier post-war survey: "Comparatively few old people live a life of complete isolation, the great majority living in contact with their children so that they have to be considered as part of a family unit rather than as separate individuals." (18)

These studies have obvious affinities with those of Gruenberg on the psychoses of the elderly in Syracuse, New York (11).

Dr. Cumming has also reminded us of Parson's functional analysis of what goes on in family life: the essential process of socialization of the younger members, and the secondary process of stabilization and control. Here I confess to some perplexity about the difference between these stages, since "stabilization and control" seems simply a continuation of the socialization process. Perhaps if I go back to read Parsons *in extenso,* I will understand better the contrast he introduces there.

Dr. Cumming finally indicated the major antitheses in roles within the family members—the contrasts in power roles between parents and children, the predominantly instrumental roles of the males, and the socio-emotional roles of the female members.

At this point I was reminded of the more elaborate breakdown of family in its process of development used by Dr. Lilli Stein (19)

in a survey in the suburbs of Edinburgh. She found it rewarding to look at families with no children, families with pre-school children, families with children going to school, families with young unmarried adults, and (as occurred frequently in the post-war situation) families with younger members who are married but living with the parent household. This type of breakdown was found important in a tuberculosis study and would be, perhaps, no less important in a study of mental disorder.

One incidental by-product of Dr. Stein's survey was the discovery that in this suburb of Edinburgh, where we tended to think that divorce and separation were relatively infrequent, families with school children had an over-all rate of 16 per cent broken homes. This is the base line against which we have got to measure the significance of broken homes in any future clinically-oriented studies in similar communities.

On page 197 of his paper, Dr. Cumming mentions in passing that he wishes there were better definitions of terms employed for studies of emotional deprivation and separation in the family.

In fact, Bowlby has filled that gap. With Mary Ainsworth, he published a monograph in which they discuss with great clarity the necessity of distinguishing between physical separation and various forms of emotional deprivation where no physical separation from the parents had taken place (1).

It was after writing this essay on methodology that Bowlby carried out his careful study of children in a tuberculosis hospital (4) at the end of which he very candidly admitted that the outcome had not been as he predicted—an admission which did more to inspire confidence in the soundness of his observations than the rather sweeping claims which preceded (and, regrettably, followed) this publication.

Dr. Cumming invited me, in discussing his paper, to draw attention to some recent family studies in Britain. Accordingly, I have reviewed a number of such studies and tried to place them in the context of his charts. This was not always easy.

The first thing I found was that it wasn't particularly rewarding to use a separate column for socialization and another for stabilization because I failed to see the conceptual contrast between the two processes. My next difficulty lay in separating studies of functional process in the family from studies exclusively concerned with struc-

ture of the family. In fact, I found that both elements were present in many of the studies I was concerned with.

For example, Bowlby, as one knows, is continuing studies of deprivation in the family setting. His earliest studies were concerned with fracture; that is, with a physical situation in which one parent —the mother—was removed from the family situation or the child was removed from the mother. His more recent studies have been concerned more with anlyses of emotional deprivation in the family scene, whether physical separation has occurred or not.

In contrast, Dr. James Douglas (who is carrying forward a cohort study of a sample of all children born in England, Wales and Scotland in one week of March, 1946) is of necessity concerned with structural aspects because his population is dispersed throughout the country and his type of information is largely documentary (supplemented at intervals by interview data).

Nevertheless he has been able to throw some interesting light on this question of separation. It is he who informed us that when a child is separated from its mother yet remains home in familiar surroundings the observations do not indicate any distress in the child; whereas if the child was removed from its mother and also taken away from home at the same time, it showed nightmares and other signs of emotional disturbance for quite a long period after that event (8).

A valuable contribution to this field of inquiry was Dr. Hilda Lewis' dispassionate assessment of the outcome, two years after passing through an experimental treatment center, of 240 children whose family life had become severely disrupted (14). This monograph differed from some other studies of childhood deprivation in the sobriety of its findings, and in drawing attention to the fact (as Skeels, and Hewitt and Jenkins have done in the U.S.A.) that certain types of parental incompetence have predictable ill-effects on their children.

Several surveys have been made of the conspicuously inefficient or "problem" families and of the condition of children who have grown up in such a disorganized environment. The findings of five extensive (as contrasted with intensive) studies of this kind have been published by the Eugenics Society, in a small book which contains a useful bibliography and a discussion of the methods used in this type of research (2). In a subsequent paper (3) the same author ana-

lyzed the frequency of disruption of marriage in the histories of patients suffering from various psychiatric illnesses and put forward a proposal for preventive action to minimize the consequences of this family breakdown.

The interaction of parents and children was the principal theme of a study by Miss E. M. Goldberg (9) which sought clues to the etiology of duodenal ulcer by contrasting the family background and childhood experiences of 32 young male patients and 32 controls. As so often in this type of inquiry, the findings were not sufficiently clear-cut to convince a skeptical reader of the ultimate importance of the types of faulty relationship which were inculpated. This can be ascribed to the author's honesty in making no concealment of the great complexity of the interactions observed.

A similar criticism can be advanced of the monograph by Elizabeth Bott which had the merit of trying to advance theoretical concepts which would permit a more sophisticated analysis of roles within contemporary middle class urban families. The attempt was a courageous one but, as a fellow sociologist pointed out in a review of this work (23), certain of her key concepts—such as the contrast between "loose" and "close knit" social networks—were insufficiently supported by data of observation.

A part of Miss Goldberg's more recent work has something in common with the studies of Lidz, Bateson, Bowen and Wynne, who are searching for etiological factors in psychogenesis of schizophrenia. However, it is only part of her work because being in Dr. Morris' unit she is obliged to temper her purely functional interest with statistical and structural inquiries. In this area, she has been collaborating with Dr. Stuart Morrison in his studies of the families of young male schizophrenics.

Morrison's work is cited by Dr. Dunham as a contribution to the debate about social drift in the life history of schizophrenics. Morrison showed that though young schizophrenics accumulate in the lowest occupation classes, their fathers a generation before were found to be evenly distributed, in terms of occupation, throughout the population of England of a generation before (16).

Miss Goldberg is following this up by studying two series of 100 consecutive young male schizophrenics, first-admissions to two large hospitals near London. She has allowed me to see a preliminary report which confirms Stuart Morrison's finding and also gives more

information about what has been going on in the schizophrenic patient's life experience. She indicates that although the patients, as a whole, were quite good at school (they were actually better than a control population in their school experience) thereafter they were non-starters in occupational terms. They never got beyond the two bottom grades in the occupational ladder.

Dr. E. H. Hare's work is referred to in two of the papers before us today. He is also interested in the life history of the male schizophrenic, and he has committed himself to the opinion that the accumulation of young male schizophrenics in the socially disorganized parts of cities is frequently a consequence of the development of the illness in their own life histories (12).

Dr. Jack Tizard and Miss Jacqueline Grad have also been interested in the family of orientation; in this case, of imbecile and idiot children living in Greater London (10). They have been concerned with the child but more especially with the family. They set out to determine the consequences both to the child and to the family if this severely handicapped child were, or were not, admitted to an institution.

Going on to the next phase of family development—"Limbo 1"—I can cite three people interested in juvenile deliquency. My justification for putting them there is that they study young people who have escaped from family control either while attending school or as members of adolescent gangs. These workers show a graduation of research interest from analysis of personal interaction—which is Dr. Peter Scott's first concern—to Dr. T. C. N. Gibbens—who combines biological, clinical and statistical approaches—through to Dr. Leslie Wilkins, whose studies have been documentary and statistical.

Dr. Mervyn Susser and Dr. Zena Stein, working in the Manchester University Department of Social Medicine, have made a study of the experiences in young adult life of boys and girls who went to schools for the mentally handicapped: young mental defectives of the higher degree.

At this stage, I should like to draw attention to the study by Professor Stengel and Miss Cook (20) on attempted suicides. One is in a quandary where to place a study like this because some of the subjects came from families of orientation and some from families of procreation. I put it here simply because they are, on the whole,

a younger age group than those that were studied by Sainsbury (17) in his monograph on suicide in London.

Sainsbury is also impossible to place accurately in any one of these pigeonholes. If I associate his study with the second type of familial limbo—that which supervenes after the family of procreation has dispersed—it is because a large proportion of his cases belonged to this older age group, and suffered from its characteristic disadvantages. An important finding of this study was to point to the correlation between residence in a single room in households broken up into small subdivisions and high rate of suicide in different boroughs of London.

An instance of research centered on the family of procreation, is found in the work of Mrs. Margaret Brandon (5). She studied mental defectives, who, after having been "ascertained" (by the Mental Health Officer of the Local Authority) or treated in institutions, had subsequently married. She was concerned to see to what extent their marriages had either succeeded or become casualities and also what the consequences were for their offspring.

Miss Enid Mills, whose study can also be listed under the rubric of the family of procreation because most of her subjects are in this category, is engaged in studying admissions to mental hospitals from Bethnal Green. An associate of Dr. Michael Young, she has been able to compare the families from which a patient was admitted to a mental hospital with the families of Bethnal Green, as a whole, on which Young's group has such excellent data.

She is comparing them in respect to structural characteristics and also behavioral characteristics, using the same demographic and behavioral observations as in the earlier study, e.g. their measures of types of interaction, such as the frequency of visits and the frequency of asking for help between different categories of kin in the families in this area.

Two of Miss Mills' preliminary observations perhaps are worth bringing to attention. First, she notes that Bethnal Green has a very stable, old-standing, East End population with one of the lowest percentages of immigrants of any part of London, and yet in her counts of the area's mental hospital population, there is a disproportionately high proportion of immigrants. Miss Mills finds in Bethnal Green that people tend to minimize the recognition of mental disorder and then suddenly go overboard and label it as

something very severe and probably irremediable. She points out that residents are slower to identify insanity or gross mental disorder in the members of families which are well known and which have high standing in the community. This perhaps contributes to the disproportionate number of immigrants among her patient population.

In our own Social Psychiatry Research Unit[1] we have been concerned, Mr. Brown, Miss Topping and myself, with a follow-up of chronic psychotic patients who leave the mental hospital and return to the community. This too can be considered in relation to the family of procreation because one of the most striking findings in our study was the contrast in outcome of schizophrenic and other types of patients, notably manic-depressive patients, in respect to the household to which they returned (6).

We found that the schizophrenics fared worst in the marital household, nearly as badly in the family of orientation (their parents' households) and very much better with distant relatives or with strangers. The opposite was the case for the affective psychotics, who fared best with their wives and worst with distant relatives and strangers.

Finally, I might refer to one or two studies concerning the later stages of life, such as that of Dr. Kenneth Rawnsley in South Wales. He is engaged in the measurement of attitudes towards chronic mental illness in the population of South Wales, and his first interviews have been with the kinsfolk of patients who have been separated from their families by long periods of hospitalization. The separation was so complete in some cases that his interview served to remind his informant that the forgotten patient was still alive.

Professor Sir Aubrey Lewis was responsible for a wartime study of social factors related to the admission of the elderly patients to mental hospitals (13). This was a modest inquiry but it indicated very clearly the need for more systematic research into those cases in which the disappearance of former family support had contributed to the elderly patients' being admitted to hospital.

In this brief conspectus of recent British studies of the family it may be observed that there is a bias towards empiricism and a relative paucity of theoretical formulations. This can, perhaps, be attributed to the influence of major trends in social policy which

[1] Social Psychiatry Research Unit, [British] Medical Research Council.

led during and immediately after the second World War to the establishment of the welfare state. F. A. E. Crew has commented (7) with characteristic pungency on the consequences for family life of these events: "Changes in the social and political structure of society have tended to take the power out of the hands of parents and place it in those of the head of state. . . Whether or not the replacement of parents by bureaucrats is a good thing must be determined by reference to the quality of the care given to the young by the two parties."

Simultaneously with the growth of public responsibility for deprived and handicapped members of the community there has been a critical reappraisal of public institutions. As Titmuss has pointed out (21), post-war Royal Commissions have condemned, in turn, large institutions for orphans, for paupers, for the infirm and elderly, and for mental patients: the emphasis has shifted to the home and the family. In psychiatry, particularly, "community care" is now the predominant theme. The passing of the Mental Health Act, 1959, with its liberal provisions for informal treatment of the mentally ill has only accelerated the demand for further studies of the circumstances and the interactions in families which contain a psychiatrically disabled member.

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SUMMARY OF DISCUSSION

1. What was implied by the idea that a family may keep its members *unduly long*? Dr. Cumming suggested in his paper: " . . . the family must remain a sub-system of the total society.

If it takes on too many of the aspects of a self-contained system, it tends to retain its members unduly long and to prepare them inadequately for membership in alternative groups."

In more general form, the question concerned the length of retention and its relation to personal and social disorder. When applying the question to the present day family in a Western urban setting, many observers stated that trouble often stemmed from too short a retention in the family—quite the opposite of "unduly long" proposed by Dr. Cumming. Sociologists like Burgess and Zimmerman at Harvard believe that the family has developed into such a specialized social sub-system, with so great a loss of former functions, that it no longer prepares the child adequately for leaving the family of orientation and living outside it as an adult. This factor is seen as contributing to an increase in some kinds of mental illness, especially neuroses and stress disorders.

To be sure, the entire question is relative to the setting in which the family is found. Each culture provides its own concept of what a family is and does, and it provides role expectations for the various members, both inside and outside the family. In Chinese and Indian cultures with an extended family system, many functions remain within the household which govern economic and religious aspects. For such cultures the same issue (incompatibility between the individual's socialization within the family and the social demands imposed upon him outside the family) does not occur.

In the United States today, recent data from the Census Bureau show that the individual spends far less time in the family of orientation than ever before. Concurrently indices of personal and social disorganization, such as rates of delinquency and divorce, are recording increases. It seemed most plausible to infer that these two sets of facts are related: that offspring leave the family of orientation not yet completely socialized and are passed on to other groups ill-equipped to continue the process, thus placing the individual in stressful situations and the likehood of consequent disorganization.

Hypothetically, either too long or too brief a period of retention in the family might produce undesirable effects. It would be a matter of empirical study to learn which situation was true of any particular setting and how much of a problem it created. Operation-

ally what is needed is to establish various indices of disorder and to relate these to length of retention, graduated into time intervals.

This information should prove very enlightening, although it can not of itself settle the problem of conceptualizing what is going on. The concept of "unduly long" implies that there is some standard by which duration can be judged. However, if the standard is defined in terms of social expectations, then there exists the danger of circular reasoning.

2. The last point suggested a similar caution—that of confounding cause with effect. As an example, one of the most widely quoted studies of maternal deprivation was by Goldfarb. Close inspection of his data reveals that at least two of the 15 cases which remained in the institution were grossly defective. Analytically, two cases are sufficient to produce statistically significant differences between the experimental and control groups. It seems most probable that the supposed *effects* (maternal deprivation through institutionalization) were actually *causes* in several cases; that some of the children were kept in the institution *because* they were either defective or damaged.

Dr. Pasamanick pointed out that the data he and his associates gathered prospectively could have led to similar confusing inferences had the same children been studied retrospectively. Among brain-damaged infants the amount of brain injury has a significant direct relationship both to the amount of tension in the mother as well as to the amount of hospitalization of the child. Thus, had they studied these cases retrospectively, they would have found that behavioral difficulties were directly related to time spent in the hospital. From which they might have inferred that maternal deprivation was the cause of the difficulties.

3. Other discussion pointed out that sample size was quite small in the great majority of studies on the family. This raised questions as to the validity and statistical significance of their findings. However, it is possible to take a broader approach, in line with classic epidemiological methods, by working with a population rather than a small series of cases. In Singapore, Dr. Murphy was able to use such an approach, one which has to date been largely neglected in the United States. What is needed is to set forth clear-cut, recognizable characteristics by which families or households differ, such as whether or not they had pre-school children.

Dr. Kramer noted that he and his associates have tried to get

mental hospitals to keep records that would yield family data comparable to the Census classifications, but with little success. Such data would be important both for studies of a retrospective and prospective nature, as well as for devising follow-up programs.

4. The treatment of families rather than the individual index case currently is an area of increasing interest. It was noted with disappointment that Dr. Cumming had not included it in his paper. It was suggested that attention to it has been long overdue because psychiatrists by concentrating on the one-to-one doctor-patient relationships have not adequately met patients' needs.

5. To the Parsonian proposition that socialization and stabilization were two basic functions of the family, a third one was suggested by Dr. Murphy's work in Singapore. The concept conveyed by the term *sharing* referred to conjoint participation in decision-making and in performance of certain tasks within a family. It is Dr. Murphy's impression that sharing plays an important part in the prevention of certain types of mental breakdown in later life.

Dr. Cumming was queried on a number of specific points in his paper.

6. The widely used terms *structure* and *function* have been viewed rather differently by different writers. "Structure" can refer either to studies with a statistical approach and involving the use of questionnaires or to a way of abstracting phenomena. Dr. Cumming's paper seemed to intend only the latter usage.

7. Similarly, the usual meaning of the term *extended family* in anthropology is the web of interrelated households spanning several generations, ranging from the Navaho system to the "kissing cousins" of Virginia. Dr. Cumming's usage, it was noted, was more limited, referring only to the addition of another generation to the household.

8. Dr. Cumming, in comparing the child raised in an institution with the child raised in a family, had, apparently, backed away from the idea that the effect of the former leads to mental disorders. His suggestion that the two modes lead to two different kinds of people brought forth the query as to whether there was any evidence for his suggestion.

9. The characterization of male roles as instrumental and female roles as emotional, in the family, was not readily understood. For example, was it affectional rather than instrumental for a mother

to make beds, or to prepare food, or to go out and work, as many did?

10. Finally, Dr. Cumming was asked to enlarge upon his remark that ". . . . organized study of the area of the family and mental illness is in a state of chaos." Was the situation due to something in the training of the people in the field? Might this be changing? What prospect was there of overcoming it?

DR. CUMMING: The words "unduly long" seem to have created some confusion. The discussion (Point No. 1) in pointing out that my presentation is culture-bound has, I think, helped me in explaining this term because the viewpoint is indeed culture-bound, and intentionally so. In our society it is generally agreed that once a male child reaches a certain age he should become self-supporting. Even if he is wealthy he is supposed to work if he wishes to avoid the epithet of "play boy." There is a value on work and on independence.

However, if we remember that there is a power differential between the generations of the nuclear family, and that the power of the father derives at least in part from his occupational role in the larger society, we can anticipate difficulties arising within families as the boy reaches the age when he is expected to work. If the boy does not work it is felt that he is not doing his part. Conversely, if he gets work and remains at home he has materially lessened the power gap between his father and himself and has created a situation of potential strife.

Thus a true dilemma can develop for the male child at a certain age. If the child remains at home after this dilemma develops, we would characterize this as being at home "unduly long."

We hope that in the near future we will be able to add data to speculation on this problem through a study in progress at our Unit.[1] This study will allow us to examine the length of time that people spend in their families of orientation and in the limbo state, and to relate this information to hospitalization for schizophrenia.

The thesis that there has been an increase in mental illness because the family has decreased in significance is an interesting one, but one about which the current evidence is ambiguous. It seems to me that we need a great deal more study of the way in which the func-

[1] Mental Health Research Unit, New York State Department of Mental Hygiene.

tions which were formerly performed in the nuclear family are accomplished in modern society. Walter Miller (in a paper which I believe is yet unpublished) suggests convincingly that the street corner gang in lower-class neighborhoods has the function of socializing the male child into an adult male role, a function which might otherwise be lacking because these families often lack an adult male member. It is true that a high price is paid here for the performance of this function. We do, however, need to examine how well these various socializing functions are performed, both within the family and within substitute socializing institutions, and the price paid in each case. Only then can we estimate whether the older form or the newer one is more advantageous to our present social system.

Thus this thesis and mine are opposed. We shall have to await experimental evidence to decide which of the views is more accurate.

I would like to agree with, and emphasize the point concerning the need for what I have in another context called "markers" for the types of family interaction (Point No. 3). In England, Elizabeth Bott has characterized two polar types of family structure which have definite interaction styles. It is obvious that her typology does not apply to American families, but I am sure we could find different types of our own if we studied family structure closely enough. If we could locate structural markers, they would make epidemiological studies much more fruitful.

Now a word on Parsons' terms, the "instrumental" and the "socio-emotional" roles (Point No. 9). Two things should be kept in mind. First, these terms must always be thought of with reference to a given system. Secondly, since all roles have both elements in them we are always speaking of a *relative* predominance. The mother who takes a job outside the home is undoubtedly playing an instrumental role, but when both mother and father are at home with their children it is likely that her role will be much more socio-emotional than his will be.

I am glad that the discussion called attention (Point No. 7) to the fact that I have given the term "extended family" a very limited meaning in this paper. I have used it to mean the addition within the household of any family member other than two generational father-mother-child complex. This meaning is obviously too

limited. While Parsons has postulated that the American family is by and large a nuclear family, studies such as those of Elaine Cumming and David Schneider have demonstrated that this is not really so. There seem to be interesting and important functions served in our society by the extended kinship group. Since these functions have been so little studied we cannot even speculate about their relationship to mental illness.

PRECIPITATING PROXIMAL FACTORS IN THE OCCURRENCE OF MENTAL DISORDERS: EPIDEMIOLOGICAL EVIDENCE

DONALD D. REID, M.D.

THE aim of this working paper is to review the evidence which has accrued from epidemiological studies on the relation of various external events or immediate environmental circumstances to the onset of mental disorders of several kinds. It is not concerned with the clinical detail of such disorders but rather with the part which the external factors may play either in increasing the risk of disease onset or in modifying its evolution. From the viewpoint of possible preventive action we are interested in the detection of a specific enviromental hazard which increases the incidence of a particular psychological disorder in a population group above the level which might be expected on the basis of that group's demographic and other characteristics. The crucial question is whether the individuals who comprise this excess in mental morbidity would have developed the disease at that point in their lives without exposure to this external stress. In other words, can the onset of disease be entirely explained by constitutional, physical or other factors, or does the external stress add appreciably to the risk of precipitation? To answer such questions with any conviction needs an equal certainty in the quantitative assessment of the external factor and of the reaction of the individual and population to it; and there has been a tendency to confuse in the single word "stress" both the apparently noxious stimulus and the presumptive response. Indeed, there are few circumstances where there might be any very wide agreement about what constitutes a potential hazard to mental health and fewer where an unequivocal assay of its potency can be proposed. It is thus hardly surprising that the epidemiological or statistical evidence about the effect of such externals on mental disease is relatively scanty and that much of it comes from studies done in war. Only in war are large segments of the male population, selected in a

fairly uniform manner, exposed to threats to life which are obvious and severe enough to be accepted as a universal emotional stimulus. Even in such relatively advantageous conditions for investigation, however, problems of interpretation soon become manifest. It thus seems sensible to set out some examples of studies done largely in the second war in both military and civil populations so that the applicability of methods and results to the less extreme circumstances of peacetime can be more fully assessed. Some examples of studies made between the wars are also discussed.

War and the Onset of Mental Disorder

The stress of war is now readily associated in the lay mind with the onset of psychological breakdown, but its effect on medical views about the nature and origin of psychoneurotic illness is perhaps less widely appreciated. Recently, Adrian (2) has described how, just as the Spanish-American War hastened the eradication of malaria and other tropical infections in that part of the world, the first World War with its outbreak of "shell-shock" cases within a few months heightened the interest in the etiology and treatment of the neuroses. At first, as the diagnostic label "shell-shock" implied, it was thought to be the result of mechanical damage to the brain—a *commotio cerebri* due to the blast of high explosive. Only later did the emotional origin of the mutisms, deafness, paraplegias, and convulsive attacks become apparent; and in this realization the still relatively novel Freudian teachings about the pathogenesis in emotional conflict, sexual or otherwise, of the neuroses doubtless had their influence. In the conditions of air fighting in the earlier years of the first World War, there were many physical factors specific to the air, such as oxygen lack to which syndromes labelled as "flying stress" could be quite reasonably attributed and Birley (5) discussed the part they might play in causing neurosis among pilots. The abnormal results in cardiovascular tests seen in patients with severely disabling neurotic symptoms served to perpetuate this notion and to

overemphasize the physical component in the causation of such disorders (13). Although the essentially emotional origin of these disorders was clearly recognized by writers such as Anderson (3), there seems to have been a lingering suggestion that neurosis in pilots, for example, was usually the cumulative result of the stresses, physical as well as psychological, inseparable from air fighting. Clinical impressions of the effect of acute episodes of fear in action rested on the time relationships between such events and the onset of symptoms, on the occasional suppression of these incidents in clinical histories and on the results of abreactive methods of treatment. On the other hand, controlled studies, where comparisons are made between the frequency of terrifying crises in the history of patients with neurosis and in healthy soldiers in the same unit, were conspicuously absent. In the first World War there seems to have been no special systematic attempt made in the Allied medical services to investigate these disorders, whether of infantrymen or pilots, by methods now classed as epidemiological. The success of treatment by abreaction would seem to have confirmed the overwhelming import of emotional conflict; but, as Adrian remarks—"without statistics, and with the fortunes of war changing from month to month, the evidence in favor of treatment by abreaction was not convincing proof of the theory behind it."

The need for statistics on the fluctuating patterns in neurotic disease incidence as a guide to the effects of war stresses was thus patent; but even in the second World War this type of evidence was not immediately sought. The development of "operational research" (i.e. the application of scientific methods to the problems of decision-making in human organization) in the British forces was, however, paralleled by similar field survey work in the medical services on the prevention of neurosis and inefficiency in combat. The methods used were in effect epidemiological; for the intention was to relate the incidence of neurosis to such measures of "stress" and "fatigue" as could be devised and to compare the incidence in men, units or forma-

tions with different operational roles and at different times.

Although reports on the mental health of the United States Army (42) during peacetime showed the importance of neurosis as a cause of hospital admission, particularly in white troops serving overseas, the impact of war was needed to produce observations on the effects of acute stress on complete military communities. A typical report comes from Hogan (24), who, as senior medical officer on board the aircraft carrier U.S.S. WASP, surveyed the officers and men of his ship some time after it had been torpedoed. He reported that although one-third of the crew had an immediate emotional reaction and were still nervous, apprehensive and "jumpy" three weeks after the attack, only two developed neuropsychiatric disorders. This experience seems to underline the need to view the problem of the effect of such events from three points of view:

1. The immediate effect of one or more presumably frightening events;
2. The cumulative effect of a series of such events over a period of operational duty;
3. The relative importance of these external circumstances compared with differences between the constitutional predisposition of individuals to neurotic illness.

The many reports of admissions to military hospitals in all theaters of war are of limited epidemiological interest because the cases are not related to any parent population. Sims (36) tried to overcome this defect by comparing the ratio of patients with anxiety and depression to those with hysteria or schizophrenia and he suggested that, in men from different army units, this ratio varied in step with the degree of initial selection, i.e. with better selection the proportion of hysterias and psychotics was low. Although he thought that the ratio might be affected by operational stresses affecting particular units he did not suggest that it could be used as a useful indicator. From the point of view of Army statistics, Appel (4) warned that very considerable fluctuations in the rates of hospital admission or discharge from the Army because of neuropsychiatric

disorder resulted from changing demands for military manpower during the course of a war. For these reasons, hospital and similar data from the services proved to be less useful in the epidemiological study of psychological disorder than might have been either hoped or anticipated. Similarly, the reports, such as those by Cochrane (8) or Tas (41), of neurotic disturbance in groups exposed to particularly distressing circumstances either as prisoners-of-war or as inmates of concentration camps are, inevitably, anecdotal rather than statistical or epidemiological in nature.

In the Royal Air Force, a system of notification of patients referred by squadron medical officers for psychiatric opinion was set up quite early in the war (40). This allowed cases to be related to the formation populations exposed to risk and the calculation of incidence rates. The first broad review of these rates showed that the highest incidence rates were in the operational commands (Bomber, Fighter and Middle East) and that the rate in the Middle East, despite the added strain which might have been attributed to separation from home, was appreciably lower than the maximum rates which were found in the United Kingdom based Bomber Command. Within the latter Command, a more detailed study was made of the factors which might account for similar variations between squadrons in the incidence of disabling neurotic illness (34). Two indices of "stress" were used: a measure of the hazard or risk that the crews had to face stated in terms of the casualty rate per cent in each particular squadron each month; and the index of the duration of the flying effort demanded from them given by the average total number of hours operational flying completed each month in each squadron. The attack rates from both neurotic and venereal disease were taken as alternative indicators of the possible effects of these external factors. A correlation analysis of the variations in time of these several indices showed that a period of heavy casualties was accompanied by a high incidence of neurotic illness in the same month in the squadron affected (and a sharp rise in the frequency in venereal

disease in the month thereafter). On the other hand, mere duration of flying effort unaccompanied by heavy casualties had no such time relation. Similar observations on the correlation of battle casualties and neurotic breakdown were made in other theaters of war (e.g. Hanson and Ranson (20) reporting on experience with the 45th U.S. Division in Italy in 1944).

The indications given by these crude correlational studies were followed up by comparing the incidence of neurotic breakdown among men at different stages of the tour of thirty sorties against German targets then current in Bomber Command of the Royal Air Force. It was found that the maximum incidence was during the initial five sorties of the tour and that after the twelfth sortie there was no further rise in the incidence curve. This finding would support the earlier suggestion that the immediate effect of operational conditions on predisposed men is greater than the cumulative stresses in those who survive the initial testing period. At the same time, it should be pointed out that the early breakdown of the more susceptible may explain at least part of the association in time between the occurrence of heavy casualties and a high incidence of neurosis. As replacements are called for during a period of heavy fighting, more inexperienced and untried men will be exposed to the operational test and an outbreak of fresh cases of neurosis is inevitable. When, however, some allowance was made for this possibility by adjusting the rates according to the proportion of operational novices in each squadron, it did not obliterate the time relation of hazard to neurotic illness.

The resultant effects of the balance between the standard of selection and the specific stresses of the individual's operational job are seen in the contrast in incidence between air gunners and pilots. The maximum rate was found in air gunners, who may have had an isolated post of duty but who were less rigorously selected either initially or by progressive elimination during training. The low rate among pilots, despite their more skilled and responsible task, presumably resulted from the degree of psychological selection to which they had been subjected

during training. Because of this composite effect of selection and the circumstances of the particular job, these occupational comparisons are of little value in assessing the part of specific operational stresses in aircrews. It is unfortunate, too, that even in the second World War, there were no reported studies of the immediate past history in carefully matched groups of patients and controls. On the other hand, there were psychiatric reviews of samples of men who had completed a tour of operations without reporting sick because of psychiatric disorder. Hastings (21), for example, showed that 95 per cent of a group of 150 such men had developed symptoms during their tour; one-third of them had more severe disturbances but never came under psychiatric care. The limitations of conclusions about the effects of the crises of war on men based only on those seen by the psychiatrist are thus patent and alternative means of assessing these effects are clearly needed. This is of some practical importance in preventive medicine among troops in combat where a tolerable limit has to be set on the duration of the operational duty required of them.

Alternative Measures of the Effects of Combat

Clinical experience of patients suffering from acute neurosis suggested that some of the physical concomitants of the illness might serve as indicators of response to stressing situations in the absense of specific complaints of an obviously psychological nature (e.g. Reid (33)). Other forms of reporting sick, e.g. for trivial upper respiratory disorders, are a frequent portent of impending breakdown; but the trend of minor sickness reporting at different stages in the tour in Bomber command did not prove to be a very sensitive indicator. Body weight, on the other hand, was found to decline significantly between men beginning their tour and those who had completed up to six sorties; it rose slightly among those who survived up to twelve sorties; and it remained constant thereafter. This was in line with the fluctuations in the incidence of neurosis at the same stages of the operation career and appears to imply that both indica-

tors of the effects of stress agreed in suggesting a marked immediate effect of the conditions encountered and a much smaller effect (on those who survived) from continued exposure to such conditions up to the then prevailing limit of thirty operational sorties. Disorders of binocular fusion was another physical measure used in this context. Crews serving in the special Pathfinder Force of the R.A.F. Bomber Command were examined after return from each of thirty sorties using the Livingston gauge of binocular vision (35). As before, there was a deterioration in the power of binocular fusion which coincided with the initial period of stress already noted. In this group, either because of the sensitivity of the technique used or the special stresses of their operational role, there was also some indication of a deterioration in the last few sorties among those who had survived the whole tour. In the Western Desert, Graham as medical officer of an armored brigade, surveyed the distribution of blood pressure among troops who had spent at least a year in desert warfare (17). He found a symptomless hypertension (diastolic pressures of 100 mg. Hg., or more) in 27 per cent of the 695 men examined; and he found it difficult to attribute this to any factor other than the emotion aroused in action. After two months' rest, most of a group of men that he re-examined were found to have a normal blood pressure. These physical measures thus agreed with the trends in psychiatric morbidity in suggesting that the emotions and anxieties of battle were producing an immediate effect which resulted in disabling neurotic illness in those more severely predisposed to it and appreciable physical changes in others who did not come under psychiatric care. Although the relatively early breakdown of those susceptibles accounted for many of the time trends in incidence, some of the variations in incidence must be attributed to variations in the intensity of these external stresses.

The Effects of War Conditions on Civilians

The outbreak of war and the aerial bombardment of Britain did not appear to cause any dramatic change in the rate of ad-

missions of psychotic patients to the country's mental hospitals. Indeed Hopkins (25) reported a decrease in admission to the observation wards of hospitals serving the Liverpool area. This could be attributed to "the increased opportunities for employment, the strengthening of the community spirit, and a lessening of that consciousness of mental isolation so favorable to the development of psychological abnormality." Hemphill (23) pointed out the particularly marked effect of the War in lowering female admissions through the provision of alternative outlets for activity.

Some of the changes in admission rates may reflect other contemporary social changes but many of them, such as the increasing difficulty of caring for the aged sick at home, were unlikely to reduce the admission rate. As Svendsen has remarked in his discussion of contemporary experience in Denmark (38), many such features of the social environment can alter the threshold for admission. Nevertheless, there was no evidence that the privations and anxieties of the War, either in the United Kingdom or in enemy-occupied countries like Denmark, had produced any major increase in serious psychosis.

The evidence about the frequency of frank neurosis, of "psychosomatic" illness or of panic states is less clear-cut and the subject has excited some discussion between Meerloo (32) who urges preparations for the psychological consequences of air attack on civilian populations and Enloe (12) who denies that past experience suggests that this is worthwhile. Lewis (29) has given the most comprehensive review of British experience in this respect. He reported that, on the basis of data from the records of a general practice in London, there was some evidence of a slight increase in the frequency of patients reporting sick with neurotic complaints often a week or ten days after the bombing. Most of these had suffered from such illnesses in the past; and anxiety and depression, rather than hysteria, were the usual modes of clinical expression. The incidence of neurotic illness was quite low in firemen and civil defense workers who were exposed to considerable dangers in the first two years

of the War. As already noted, neither independent observers nor hospital statistics suggested any major rise in the admission rate to mental hospitals, although, as the War went on and shortages in housing, clothing, and food became worse, many senile dements, who would normally have been cared for at home, had to be admitted to hospital. In Scotland, where data on suicide were available, the death rate from this cause fell. Juvenile delinquency increased and there was a considerable rise in road and industrial accidents. Lewis raised the possibility that these events reflected the same environmental factors as conduce to neurosis.

Broad surveys such as that of Lewis were supplemented by more intensive studies of particular aspects of civilian reaction to bombing and other hazards of war. Fraser *et al.* (14), for example, followed up a series of individuals who had been admitted to First Aid Posts after being buried in bombed houses. Of those who had been buried for over an hour, 66 per cent developed neurotic symptoms and 40 per cent a neurosis severe enough to cause absence from work; in about equal proportions, the neurosis was either temporary or persisted for at least ten months. Persistent disturbances were not confined to those with previous neurotic illness and social complications such as the loss of a cherished home appeared to delay recovery.

Some studies of the effects of war on apparently physical diseases reflected in their results a psychological element in the origin of the disorders concerned. During the intensive bombing of London and Bristol, Stewart and Winser (37) showed that a sharp rise in the frequency of peptic ulcer perforations coincided with air raids. Later, Illingworth and others (27) in Western Scotland confirmed this general time relation but remarked that the rise in the perforations admission rate antedated the raids on Clydeside and might thus be due to anticipatory tension and anxiety rather than to the immediate stress of the attack. As in the military studies, then, the immediate effects of war conditions on neurosis and physical disease often considered to be aggravated by psychological stress were ap-

parent in the civil population. But the negligible effect on the frequency of psychosis was perhaps the more remarkable finding.

Occupational and Economic Circumstances and Psychological Disorder

There are fewer obvious opportunities for the study of the effect of acute emotional stresses in peacetime civilian life. Only a few occupations, such as those of air line pilot or coal miner, would appear to present the risks which might be unequivocally accepted as likely to cause even transient emotional disturbance of the severity common in war. The comparison of the mental disorder disease experience of either of these occupational groups with those of other men is open to the objection that they are selected both from the point of view of self-selection or by initial medical examination and subsequently through the attrition due to physical disability inevitable in such exacting jobs. In the same way it is difficult to attribute to the depressing circumstances of their homes, the particular mental disease experience of the poor without considering whether the disease preceded the poverty and the poverty resulted in the living conditions. Hyde and Kingsley (26) for example, have found that the rejection rates for psychosis in conscripts for the United States Army were related both to the socio-economic level and the population density of the area from which they came; but they also pointed out that these findings could be explained by the gravitation of the mentally less fit into the poorer communities. The problem of migration, either within the social class system or geographically within town or state, is discussed in other papers; but the complications that these selective factors entail explains why most of the epidemiological papers dealing with the possible effects of stresses are concerned largely with trends over a period of time *within* large occupational or other large population groups.

Successive generations have cherished the notion that the ever-quickening pace of their lives and the stress it involves

were certainly producing a rising tide in the incidence of mental disease. By a careful review of the statistics for mental hospital admissions in the United States, Goldhamer and Marshall (16) showed that if the changing age-structure of the population were taken into account there was, in fact, no evidence of a long-term increase in the incidence of psychosis in early or middle life during the whole of the previous century. Other studies have covered a shorter period and concentrated on the relation of indices of mental ill health to economic or other circumstances which might be expected to be emotionally disturbing. Dayton (9), for example, reviewed the rate of admission to mental hospitals in Massachusetts over the years 1917–1933—a period which covered both America's participation in the first World War and the nadir of the economic depression. The War produced no demonstrable effect on the admission rate for psychosis but there was some increase in admissions of older patients suffering from senile or arteriosclerotic brain damage. Significantly, the pattern observed resembled that found in wartime Britain in that there was certainly no increase in the admission rate for the diseases of the younger age-groups such as schizophrenia and manic-depressive psychosis. The economic depression of 1932–1933 accentuated this tendency for the increasingly frequent admission of senile patients but it seems likely that, again as in England during the last War, this might reflect the domestic difficulties and disturbances which make the home care of old people impracticable rather than any real rise in the incidence of the disease. Komora and Clark (28) confirmed that the depression had produced no remarkable increase in mental hospital admission rates in the United States despite the fact that in many of the patients coming for admission the economic situation was given as a precipitating factor in the illness. Broadly speaking, however, there seems to have been little disturbance of the general trends in admission rates over the present century which could be attributed to the Depression.

Before dismissing the possibility that peacetime stresses such

as unemployment, poverty, and insecurity have any effects on mental health, it should be emphasized, that as in wartime, hospital admissions for the endogenous mental disorders of younger people may give only part of the picture. Suicide, as an index of the frequency of the reactive depression which so frequently underlies it, is an act which need cause no rise in the hospital admission rate. But it is routinely recorded in the national vital statistics. Dublin and Bunzel (11) found an inverse correlation between the suicide rate and a business index derived from the activity of a public utility corporation in the United States; and the corrrelation was particularly close for white males. Swinscow (39) has repeated this type of investigation by relating the suicide rate for males and females in England and Wales over the period 1923–1947 to the proportion of men unemployed in the corresponding year. He showed that, for males at least, there was a very close relationship between the two. The crucial point, it would seem, is that this correlation is appreciable only for males in both countries.

Unfortunately, there are few numerical accounts of the experience of the lesser degrees of mental disturbance during these critical peacetime years. Perhaps Halliday's (19) discussion of the interrelation between the death rates from diseases often labelled as "psychosomatic" and other biological indices of mental malaise is the most stimulating. He used measures of reproductive behavior such as the fertility rates and the evidence about morbidity of all kinds that accrued from the statistics of the sickness insurance scheme then in force in Britain. He also cites Gafafer's (10) data on industrial absenteeism in the United States to support his contention that there has been in both countries a rise in the incidence of disorders such as peptic ulcer which may be affected by emotional disturbance. From the epidemiological viewpoint, Halliday remarks on certain characteristics of disease behavior which he believes to signal a psychological origin: a shift in the peak incidence to the younger age groups, an increasing male : female ratio and temporal, social, and occupational variations in disease incidence.

In the "Chronic Register" of the sick in Scotland who had been continuously incapacitated for more than twelve months, there was a disproportionate increase in some causes of chronic illness between 1931–1932 and 1935–1936 when Scotland was, economically, "a distressed area." Not only was this increase more prominent for disorders such as "peptic ulcer," "gastritis," and "nervous debility," but the increase was most marked among men under the age of 55. It is possible, of course, that such increases are more apparent than real; for, under the social insurance scheme, there was a financial incentive towards being classed as "sick" rather than "fit but unemployed." On the other hand, the younger age groups are notoriously the most sensitive to changes in industrial morale. Further, there were concurrent changes in other biological indicators of social or psychological disharmony. The suicide rate was increasing while the fertility rate fell particularly among occupational groups such as miners and textile workers whose industries were among those worst affected by the depression. The male : female ratio, whether in suicide or peptic ulcer mortality, rose and Halliday proposed that all these features of community disease stemmed from some deep seated psychological malaise.

Some of these manifestations may, of course, have simpler explanations. Cigarette smoking among younger males may be the more immediate physical cause of the high male : female ratio in peptic ulcer mortality. Errors in registration of occupation at time of death, particularly among the mining population, may inflate the reported death rate among them (22). Some of the excessive morbidity among textile workers may be the result of the efflux of the fitter men from a dying industry. But the consistency of some of the evidence of the results of depression and lack of confidence in the future of which the high suicide and low reproductive rates may be expressions suggest that the adverse economic and social stresses of the thirties may well have been responsible for a real rise in the incidence of neurotic illness.

There appear to have been surprisingly few studies in peace-

time industry quite like those described in flying crews. The short-term effect of prolonged driving on fatigue as measured by psychological tests and symptoms has been investigated on inter-state truck drivers, but less is available on the cumulative effect on health, mental or physical, of prolonged exposure to such conditions. In air line pilots, McFarland (31) reported that illness rates in general bore no direct relation to the hours of flying duty until the average flying time exceeded 90 hours per month. Although done in wartime, the findings of Russell Fraser's (15) survey of the incidence of neurosis among factory workers are quite generally applicable. He reported that a high incidence of neurotic illness was associated with the performance of over 75 hours of industrial duty per week. Many of the other features of the industrial environment which he showed to be linked with neurosis, e.g. boring tasks or undue responsibility, are those which Cameron (7) has listed as presenting a hazard to mental health in industry. There is, unfortunately, a dearth of epidemiological data on the psychiatric consequences, rather than the effects on production, of industrial conditions, methods of man-management and group morale. That such features of industrial life may be relevant to mental health is implicit in the results of studies such as those of Brodman *et al.* (6). They showed that there were consistent differences over a period of time between the twenty-four departments of a mail order firm in indices such as the incidence of long and short term absences for medical reasons, the number of reports to the medical department and the frequency of unexcused lateness. Those departments where unexcused lateness was frequent also had a high incidence of absences of short duration. The authors believe that both were indicators of defective morale and that an individual's behavior in these respects was markedly influenced by the group to which he belonged. The question implicit in this result is whether group morale as thus defined materially affects the incidence of psychological disorder.

The notion of "socially-shared psychopathology" has been fully discussed by Gruenberg (18) who recounts episodes rang-

ing from the dancing manias of the Middle Ages to the "Men from Mars" panic in 1938. Certainly, these "epidemics" of disordered thought or behavior strongly suggest the infectious nature of neurosis in closed communities. Studies such as those fostered by the Acton Trust (1) have suggested that the size of the working group (or the method of its supervision) was related to the frequency of events which are presumptive indices of psychological malaise. Industrial accidents and absenteeism, for example, tended to be consistently related to the size of the working group; and this relationship has been found in various industrial and other occupational groups. Some of the excessive sickness absence rates found in the staffs of the larger offices of the British postal services, however, may be at least partly respiratory in origin; and since larger offices tend to be in larger towns, an excess in respiratory affections is to be expected. Again, as Liddell and May (30) have shown in the mining industry in Britain, much of the apparent increase in accidents in the larger collieries can be explained by a more complete reporting of less serious accidents in these larger units. Nevertheless, the mere fact of such differing thresholds of reporting suggests differences in psychological climate which may be more directly relevant to the incidence of neurotic illness than at present appears. On the basis of the evidence at present available, however, the question must still lie open.

Conclusion

It will be obvious from this account of the patchwork of epidemiological studies reported in English in the literature that there has been no sustained systematic approach to the problem of the precipitating factors in psychological disorders of various kinds. On the other hand, there are some consistent threads appearing in different circumstances which give some support to some general conclusions. On both sides of the Atlantic, and in both Wars, the stresses of civilian life appear to have had no major effect on the frequency of the psychoses of early and middle life. Economic depression has also had a negli-

gible effect on the admission rate to hospital for such conditions, although the coincident difficulties of home care, either in depression or in war, increased the rates of admission for senile conditions.

In war, it proved possible to show the immediate responses of men to a stressful environment both in terms of an increase in neurotic illness and of bodily changes expressed as averages in groups of men. While it is true that predisposition to neurotic illness may be the dominant element in the determination of breakdown under stress, the experience of war, both in military and civilian life, suggests that such stress will, if severe enough, precipitate neurotic illness even in groups of highly selected individuals.

No exact parallel can be drawn between the results of the acute emergencies of war and the less obvious stresses of industrial life in peacetime. The evidence for any direct cause and effect relationship between normal industrial conditions and neurotic illness is slight; although there are hints that long hours of boring or highly skilled work, for example, are conducive to mental ill health. In the harsh conditions of an economic crisis, on the other hand, there is good reason to believe that, while the incidence of the psychoses may be unaffected, reactive depression, often ending in suicide, and a whole group of neurotic and "psychosomatic" disorders may cause increasing disability among the men affected by the slump in their industry. Recently, interest has centered on the effect of group size and organization on indices of industrial morale which may, in turn, be related to the risk of neurotic illness among members of the group. The epidemiological study of the effect of the stresses presumed to exist within our complex industrial society is still, however, in its infancy and no firm judgment is yet possible.

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Discussion

Dr. Paul Lemkau: Dr. Reid's excellent and comprehensive study is a definite challenge for all of us to attempt a more clear statement of our hypotheses regarding etiological factors in the psychoses and neurotic disorders.

The apparent fact, reiterated and well-supported by Dr. Reid, that psychotic disorders, in the younger adult period at least, do not change in incidence under the influence of immediate changes in environment is now so well-documented that we must accept it. It would appear that we are forced to either one of two general hypotheses about the etiology of the largest group of diseases in the conglomerate, schizophrenic disease or reaction. First, that the etiology lies in some disorder established psychodynamically quite early in life, before late adolescence at least. Or second, that it springs from some genetically distributed risk factor that operates independent of the environment as the individual matures. A third, alternative hypothesis, the combination of the two, has been the refuge of all serious investigators including the geneticist, whose data do not always fit his mathematics, the difference being accounted for in the term "penetrance."

The first hypothesis, i.e., an early psychodynamic etiology, has been supported mainly in the work of Tietze and Lidz, as has been reviewed by Dr. Cumming. Unfortunately, this hypothesis is notoriously unsatisfactory from an epidemiological point of view. It is extremely difficult to explain how schizophrenogenic households

should occur equally frequently in the various markedly different cultures in which the incidence rate of schizophrenia appears similar. (I hasten to add that Dr. Reid did not say that the rate is similar in different cultures, but that I am impressed with the evidence that it is.) The second hypothesis, a genetic-biological or physiological one acting relatively independently of the environment is not a very popular one in the United States but seems to me to be amassing a strong body of evidence in its favor, albeit all indirect and circumstantial. I do not mean to rule out the third possibility—that of two or more factors working together—but I must admit to a personal proclivity to think in terms of the much despised "either or" in preference to the more generally accepted "both." The latter seems to me to offer too easy an escape from the real dilemma we are faced with. For clinical work we are forced to the "both," but in the laboratory or at the *schreibtisch* the "either or" would appear to offer the most stimulation to new and productive work. In any case, the bias of the investigator is likely to insure what is close to an "either or" attitude.

From Reid's summary it is clear that when environmental stress is severe enough various breakdowns do occur, but that there are also various degrees of susceptibility. According to Stauffer's reports, vulnerability in military personnel correlates to some extent with occurrence of nervous breakdown in the family of the subject, with previous illness experience as reported by the subject, and with extent of heterosexual social contacts.

The problem of relating the extreme military stress situations to the occurrence of neurotic and other reactions in civilian life is well stated by Dr. Reid. The problem is of the same type as that of relating the fairly well accepted statement that extreme lack of stimulation in infancy or young childhood results in a damaged personality, to the question of whether lesser degrees of isolation produce partial or less severe damage.

The problem is, it seems to me, related still further to the study of the etiology of disease in general. It is probably safe to say that every disease is multiply-caused. The diseases in which we assume we "know the cause" are those in which there is one overwhelming cause acting at a particular moment; many have pointed out that in many diseases (such as in tuberculosis in the past when positive tuberculin reactions were more common than they are now) the

"cause" was much more prevalent than the disease. A better example might be found in pneumococcus pneumonia. At the moment the disease becomes apparent, the one overwhelming cause is the one that can be acted against, so that, as Dubos says so often and so well, it is all too frequently assumed to be *the* cause even though multiple causes are easily found, if looked for.

Is not the situation very similar in combat neurosis? A single stress becomes *the* cause since it is particularly severe at the moment and there is something that can be done directly to relieve it. As Dr. Reid has demonstrated, we actually may have better notions of the other causes over and above the overwhelming one in combat neurosis, such as sleep and food deprivation, than we have in many other diseases where, beyond such crude measures as social class, there is little clear epidemiology at all. The issue might be stated that whereas the data we have shows that stress may well be the general cause of neurosis—much as external invaders are of pneumonia—some kinds of stress will cause the illness more regularly and predictably than will others, combat experience being one of the most pathogenic. On the other hand, neurosis appears also to occur when this particular pathogen is not present as *the* overwhelming cause; the analogy is far from perfect, but perhaps it will help clarify our thinking.

The problem of the relation of the morale of groups to individual vulnerability tempts me into analogies from the field of physics; into the kind of energetics that Freud spoke of in discussions of libido and which creep into many statements to the effect that "everyone has a breaking point." The problem is the shift of the critical points at which stress breaks through the "strength" of the personality in some almost physical way. Such analogical thinking is helpful in envisaging our problems but it is, of course, dangerous if it beguiles us into thinking that we are talking about what really happens. We cannot fully describe the actuality and we must keep continually in mind that we are thinking in terms of models, not the real thing.

SUMMARY OF DISCUSSION

1. Some types of proximal or precipitating factors in mental disorder were added to those listed by Dr. Reid in his paper.

Childbirth, whether as a biological or as a psychological factor, had to be considered in connection with purperal psychosis. In addition to loss of spouse, other significant losses, such as loss of child, or loss of a limb, were mentioned.

In the past, eye operations were frequently followed by delirium, due partly to drugs but also to therapeutic measures. The situation was aggravated by the procedure of keeping both eyes covered-up for very long periods of time. Today's procedure of allowing sight to the unaffected eye as soon as possible after the operation has resulted in reducing the number of delirious reactions.

It was suggested that festivities are generally accompanied by a rise in suicides and admissions to hospitals. However, it was reported that the Chinese New Year had not produced any noticeable effect upon mental hospital admissions in Singapore.

That disasters had varied implications as proximal factors was shown by recent sociological studies in the United States. On a broader canvas, various types of psychological climates have arisen. One example was the belief that the world was coming to an end. Studies as to whether these psychological climates were related to various types of mental illness might prove interesting.

It was noted that compensation neurosis had an entire literature in Germany which, though full of pitfalls, might repay study. Acute infections, such as malaria, also needed to be considered in the present context.

2. It was remarked that the discussion was focussing primarily on the type of stimulus that induced stress. In studying communicable disease, however, less attention is paid to the stimulus than to the epidemiology of the response—the illness itself—which is then related to multiple factors (e.g. tuberculosis). Have there been any studies on the physiological response to psychological stress?

3. Yet it is not the mere occurrence of a stressful stimulus that is presumably related to the development of neurotic or psychotic symptoms, but rather the individual's response to the stimulus. It follows that psychological stress has to be studied in connection with what the stress means to the particular individual concerned. Most persons would find a bombing attack to be stressful, but some persons might find the loss of a job to be even more stressful. From the psychological standpoint it could be asked how a stressful stimulus worked upon an individual. Knowledge of the psychological mecha-

nism would help in taking into account stimuli that were not obviously stressful or not easily described.

Highly relevant sociological questions also needed to be asked. For any given situation there existed individual differences in susceptibility to stress. Yet the distribution of differential susceptibility had been grossly ignored in psychiatric research. One such area was the life cycle.

3. In addition, differences in response might be related to cultural factors; or, more specifically, to a special subculture that might arise in a particular setting. Thus Dr. Reid discussed the high breakdown rates of air gunners in contrast to the low rates among pilots arguing that the difference was attributable to selection processes which placed a man where he was needed rather than upon his psychological fitness for the job. Also relevant, it was suggested, was the fact that in the early days of the War there was a comparatively low rate of survival of air gunners; which in turn produced a mythology of the hazards of the job which was communicated back into basic training.

4. Extension of the discussion of proximal factors to such matters as perception and social expectancies, led to consideration of a larger framework in which examination of the separate pieces might presumably be fitted together into a model.

The term *model* had become a fashionable word which, today, frequently replaced what was formerly called *concept* or *theory*. It was stressed that a model, as a construct of the mind, was distinct from what really happened. But, it was argued, there always existed something behind the model, in whatever sense the term was used.

Unfortunately, investigators had a tendency to reach for the complex model even when a simple explanation was at hand. While this seemed to be done consistently, there was no merit in avoiding simple explanations. Thus it was pointed out that difference in the breakdown rates of gunners and pilots might readily be explained by the fact that these two groups were drawn from different social classes. Other armed services data on social class as a predictor of breakdown existed which apparently indicated that to show symptoms or to break down was much less acceptable to the middle classes than to the lower. Again, Dr. Reid's analysis in psychological terms of the considerable rise in road and industrial accidents in England during

the War might be more simply and plausibly explained by the increased pressure for production, poor lighting, worn out equipment, and so on.

Further, it was noted that some of the things that were generally believed to be stressful needed no explanation, because upon individual examination they proved not to be stressful. Thus it was reported that a recent study on the effects of hysterectomy indicated that, where the operation was medically indicated, the subsequent psychological effects were good rather than bad.

5. It was suggested that a promising area for exploration, one in which little work had been done, was that of counter-balancing factors—factors which served to neutralize sources of stress. The studies made by Jules Coleman in the Pacific were cited as an example. Here the evidence seemed to show that the pre-combat morale of a unit was a good predictor of the rates of psychoneurotic and psychotic breakdown during and after combat.

6. The interpretation of hospitalization rates were discussed. It was observed, unhappily, that the index of admissions to mental hospitals were used as frequently to validate a point as they were dismissed as having very little meaning. Dr. Reid's belief that neither the War nor the depression had had a significant effect on admission rates for psychosis was questioned. Clear seasonal fluctuations in rates were observable, with total release rates reaching a peak around Christmas and dropping off in January. Correspondingly, admission rates dropped off in the holiday season. This did not refute the point expressed earlier that times of festivity might precipitate mental disorder; rather the rates reflected the family's desire to have a hospitalized member home for Christmas. It was suggested that the effects of these holiday releases on the patient and also on the family might be interesting to study.

The increase in first admissions in the United States between 1941 and 1945 for males between 20 to 35 (but not for any other age-sex categories) was to be contrasted with the general decline experienced in Europe where a depressive effect on rates might have occurred through disruption of the usual processes that brought about hospitalization. It was conceivable that persons were precipitated into psychosis by the War at an increased rate but were left floating around in the communities unhospitalized. It was clear, however, that this hypothesis would not apply to the military services, where

arrangements for moving sick persons to hospitals remained highly efficient.

In the United States the fact that the military acted as a case-finding agency, sending many patients on discharge from service directly to state and Veterans hospitals could explain the increased male admission rate cited earlier.

In any interpretation of wartime rates of admission, it was important to take note of the population at risk. Rates had been developed which included in the denominator all of the armed forces, or all those not overseas, or none of the armed forces. The resulting variations showed how sensitive these rates were to differences in definition.

A final *caveat* suggested that the circumstances of civilian populations in wartime varied so greatly that comparison of admission rates between the United States and most European countries might not be valid.

As to the effects of the depression, the few long-term series on United States admissions showed continuing increases in rates for all admissions between 1930 and 1937. If the rates of admission for persons without psychosis were to be considered separately, it would be found that they increased markedly from 1933 on. (These same figures showed approximately similar rates for psychosis before and after 1933). This non-psychotic rate increase might in part be a reflection of the marked development of various social agencies and services which took place during the same period. The agencies, seemingly, solved many problems by committing clients even when psychoses were not present. A close and careful analysis of existing data was needed before these negative conclusions could be accepted.

An interesting note on the occurrence of schizophrenia in the United States Navy was reported. The rate has been so nearly constant over the past 30 to 50 years that the Navy no longer publishes case figures since they would reveal the size of its forces.

7. The remark that an exclusively genetic theory of the etiology of schizophrenia was a satisfying one, if only for its heuristic value, brought forth the suggestion that schizophrenia might be divided into two types, one explainable by an exclusively genetic theory, and another for which a mixture of genetic and environmental etiology seemed necessary. The latter idea has been expressed in a number of different studies as the concept of a nuclear schizophrenia, a con-

dition that was separable from the main undifferentiated mass of schizophrenia.

Were schizophrenia exclusively genetic in origin, one might expect no difficulty in tracing the different diagnostic types by pedigree. But, as Dr. Böök pointed out in his paper, attempts to do so had been unsuccessful. The reason might be that the types of schizophrenia were interchangeable, that in one family there might occur, for example, both catatonic and paranoid types.

It was emphasized that the data on schizophrenia indicated a very considerable genetic interaction with the environment, with the geneticist thinking of the disease as a sort of reaction that might occur in many different situations from many different causes—one of the important ones being genetic. As every psychiatrist realizes, a perfectly good schizophrenic syndrome would not be diagnosed as schizophrenia until the psychiatrist could obtain the case history and could observe the patient for some time.

Genetics and the environment were always in interaction. Any consideration of the genetic factor required it to be seen as expressing itself within a particular environment at a particular moment, yet at the same time the environmental factor had to be viewed as being itself subject to change. The effect of environmental changes on cases of sickle cell traits was used as an example. While they were well on the ground, at 2,000 feet they might go into hemolytic crisis. There was no change in their genes, but they were sick or well according to the environment. Certainly social and cultural differences between environments and changes in these environments would produce considerable variation in the picture of general morbidity and mortality. Thus there was no good evidence that there were cases of schizophrenia in which the process was initiated by genes and carried further by genes without any important interaction with the environment.

These remarks were meant, perhaps, to emphasize the need for more concentration of research on the biological side. It was indeed true that genetic explanations of some psychiatric disorders was not very popular amongst psychiatrists, despite such clear examples as phenylketonuria and galactosemia causing mental defect in children. Yet another, and older, example of the role of genetics was diabetes, a condition that had been treated for decades. Still, many psychiatrists continue to feel uncomfortable about a genetic explanation.

DR. REID: I am very interested in what was said because I find this a difficult subject and I would like to take, in sequence, the various points that were raised.

First of all, I think Dr. Lemkau and I are probably the unwelcome supporters of Dr. Böök, for as far as this material goes, it certainly would come down on what I took to be Dr. Böök's side of the fence rather than on the side of the psychoanalyst. (Point 7.)

In reply to the question about physiological responses to stress and whether there have been any other studies done on this (Point 2): I am very interested in this myself because it is something I was concerned with during the War. In fact, one of the first indications I had was from a military tailor who used to come around and measure the pilots for uniforms. He made the comment that men's measurements did change quite perceptibly during operational tours of duty and this was one of the reasons why we embarked on the studies of weight loss in pilots. I have no knowledge of corresponding civilian work. I take it that the difficulty is the inability to find in a civilian context the very severe type of stress that men in the U.S. 8th Air Force or our own Bomber Command, for example, had to sustain in wartime.

I was particularly interested in the discussion on perception and social expectancies as related to the mythology of hazardous assignments in wartime in both the RAF and RCAF (Point 3). However, I think that the explanation for some of the differences in behavior under stress between various categories of aircrew was put into proper focus when it was emphasized that the air gunner, generally speaking, was not drawn from the British upper and middle classes. His appointment was not the result of a special selection procedure but rather the end point in a process of attrition whereby people who were selected under what was called the pilot-navigator-scheme, bomber-aimer-scheme, but who didn't make the grade, very often found themselves as air gunners.

This "stiff upper lip" concept of the middle classes who will not admit to the symptoms of neurosis reminds me of the comment by R. D. Gillespie in his book where he says that the reason for the low neurosis rate among the Scottish battalions in the first World War was that everybody in Scotland knew everybody else; and if a man broke down in combat, the whole glen knew about it and he was disgraced forever afterwards.

I agree, of course, on the importance of the perception of risk as opposed to actual physical risk. There was no question but that at the beginning of the War people felt that the exposed, isolated, and rather obviously dangerous position of the air gunner would have some effect on morale and neurosis. I doubt whether *in fact* this was so: quite often when a bomber crashed the one man to get away was the air gunner because the tail broke off; he was thrown clear and very frequently survived. So that as far as accidents were concerned, the actual risk to air gunners was rather less than it was for other members of the crew.

This perception of risk was also the likely explanation of the very interesting time correlation between the incidence of breakdown and venereal disease after a burst of heavy casualties.

What happened was that men perceived that life was going to be very short and, therefore, that it should be very merry. Thus the reason for time correlations with venereal disease: London was only 100 miles from the bomber bases.

One or two suggestions were made about stresses which might have been included (Point 1.). Both puerperal psychosis and delirium following eye operations are certainly mentioned in the literature, but the difficulty about these observations is that they are anecdotal rather than epidemiological in any strict sense, and I haven't included them partly for that reason.

The loss of spouse and child I did not cover because I think this properly comes within the family studies. I could find no evidence about the effect of festivities on suicides or anything else. The only time correlation of this nature that I know of is in a study showing the high incidence of perforation of ulcers in Glasgow around Christmas which presumably results from the stress on us Scots of giving each other Christmas presents!

The compensation neurosis is, as has been pointed out, an area fraught with all kinds of difficulties, and I know of no epidemiological work which has gone on in this field. As far as I know, both the German literature and our own consist of clinical accounts of cases—of the observations of time correlations between accident and onset, and compensation and recovery. It seems to me that they are rather uncontrolled observations which really should not come into the strict definition of epidemiology.

As to counterbalancing factors (Point 5). This, as far as I know,

is something that has not been much studied in any numerical way, even in the military context. I think that the assessments which were made by psychiatrists of morale were made on a rather intuitive basis, and I don't think there are any studies where there is sound epidemiological and statistical evidence of the presence of counterbalancing factors.

SOCIAL STRUCTURES AND MENTAL DISORDERS: COMPETING HYPOTHESES OF EXPLANATION

H. WARREN DUNHAM, PH.D.

THE central focus in this paper will be to examine critically the kinds of hypotheses that have been advanced to explain the findings of some epidemiological and ecological studies of mental disease. In order to accomplish this task it is necessary to point to: (1) the differences between epidemiological and ecological studies; (2) the various theoretical considerations directing these studies; and (3) the value and purpose of both kinds of studies in understanding some of the problems associated with mental health and disease.

As I have reflected on this problem it seemed to me that some clarification of the task will result by pointing to the distinguishing features of epidemiological and ecological studies of mental illness.[1] Epidemiology has been defined as "the sum of what is known regarding epidemics." (8) As such, one most important phase of an epidemiological study is to find out the incidence and prevalence of a disease in a circumscribed community setting or a carefully delimited population group. The general objective in these studies has been primarily an overall count of cases. Furthermore, most of the epidemiological studies, particularly the European ones, have been conducted by medically trained persons who have regarded their efforts as having some genetic significance. Thus, when they compare the results of their surveys they think that their theoretical position is supported when the statistical differences are at a minimum. For example, the two well-known surveys in this country, the Baltimore (6A, B) and the Williamson County, Tennessee, (39), reported rates for active and inactive cases of 62.0 and 69.4 per 1,000 population respectively. Such results frequently have been interpreted that regardless of the type of area—

[1] While in the work of epidemiologists and ecologists there is much overlapping of interests and technique, I have elaborated this distinction, even though it may not be acceptable to some, with the express purpose of sharpening up existing disagreements and expressed viewpoints.

whether urban-north or rural-south—the frequency of mental and behavioral disturbances in a delimited population is about the same.

However, epidemiological comparisons in the hands of social scientists may produce a different emphasis. Thus, Eaton and Weil (9a) using a standard expectancy method, compared ten surveys relative to the amount of enumerated psychoses. The Hutterites, serving as a norm, had a higher enumerated rate of psychoses than seven of the ten populations upon which they reported. They regard the high Hutterite expectancy ratio as a function of their thoroughness in screening and think that five of the populations ranking below the Hutterites were not screened thoroughly for their recovered mental cases. After several other arguments they conclude that the Hutterites have the lowest frequency of psychoses among the four rural populations but higher than the frequency found by Tsung-yi Lin in his survey of Formosa (28). They then point to the limitations of the epidemiological method with its capacity for showing the presence or absence of quantitative differences but without being able to explain them. "Their genetic composition, physical health, psychological tendencies would have to be analyzed in detail with the hope of identifying some patterned interrelationships that could account for the quantitative differences." (9b)

Even so, undeterred the investigators proceed to show that a sociological orientation is useful in accounting for the predominance of the manic-depressive psychosis (73.6 per cent of all persons diagnosed as psychotic among the Hutterites). After convincing themselves that their diagnostic judgments were valid, they develop the role of the "social cohesion factor." From examining various bits of evidence they conclude that the ratio of manic-depressives to schizophrenics varied in different population groups and this fact (they call it an assumption)"—would support the general theory that sociological factors play an important role in the way functional mental disorders are manifested in patients." (9c) They conclude tentatively, "The

extreme cohesiveness of the Hutterites which may contribute to their low frequency of schizophrenia, may be significant for the relatively high proportion of manic-depressive reaction among psychotic members of the sect." (9d)

I have used Eaton and Weil to illustrate the fashion in which the orientation of the investigators enters into an interpretation and also to illustrate the gap between a theory and the evidence to support it. (Weil, of course, is a psychiatrist.) That they should fasten upon the differential frequency of these two functional diagnoses among the Hutterites to show the relevance of sociological factors is incredibly naive considering the seemingly interminable problems in making the differential diagnoses and considering also that many different psychiatrists were involved in making the differential diagnoses in the other studies.

It might have proven just as valuable to make something of the fact that Bremer's Arctic Norwegian village (1a) had a frequency of psychoses that was twice the Hutterite rate, for in both studies the screening was supposed to be excellent and the data were gathered in extremely isolated communities. If we assume this difference to be a real one, then one is forced to conclude that the population of the Norwegian village is genetically inferior to the Hutterites or that the conditions of life provide a more telling impact upon the people of the Norwegian village than a different set of life conditions do in the case of the Hutterites. And this in a final sense is the big factual question—"Can such surveys as counts of prevalence and incidence of total psychopathy or particular disorders show significant quantitative variations between different cultural groups and/or different strata or categories of population within the same society?"

Now, let us turn to consider the ecological studies of mental illness. Ecology has been defined as "the science of organisms as affected by the factors of their environment" or "the study of the environment and life history of organisms." (8) This means that an investigator who wants to make an ecological

study of mental disease will be concerned in locating the mentally diseased person in some position within a culture or social system and viewing him in relation to the positions occupied by other mentally diseased persons and the non-mentally diseased in that system. Thus, an investigator making an ecological study of mental disease, or a mental disease, is concerned with discovering if the incidence of disease, or a given disease, will vary significantly between different temporal, spatial or social environments. These environments are frequently delimited as social classes, religious groups, occupations, types of families, types of communities, levels of education, historical time, or special environments such as school, military unit, or prison.

Now, if the ecologist can show conclusively that certain significant rate differentials for mental disease exist between different positions in one of these environments, he faces the task of trying to find some hypothesis derived from a theory that may explain the difference. Thus, most of these studies have been largely empirical in character and if any theory is pertinent, it is of the broadest and most general variety.

However, the ecologist focusing on the organism in his environment will be concerned with trying to isolate certain environmental factors that will explain the rate differences and that will account for the development of the disease in the person. If he operates as an ecologist he will emphasize the processes within the environment and attempt to show the social variable or complex of variables that is associated with the rate differential. If he tries to get at the social factors that are causative or predisposing for persons in that environment, he will be thrown on another level of analysis where his ecological findings will prove only indicative of some factors that he might study as having an etiological significance.

The difficulties and pitfalls that beset the investigator in this area as he strives to make sense out of his findings are well illustrated by Frumkin's study of occupations and mental disorder (13a). He begins by emphasizing that research is needed to investigate the etiology of mental illness. His data comprised

all first admissions to Ohio State's prolonged-care mental hospitals for the year 1950, who had an occupation, and who had not been classified as housewife, student, or unknown occupation prior to admission. These data (1,192 males and 347 females) were then broken down by occupation in relation to age, sex, and mental disorder. His basic findings consist of rates per 100,000 for each sex for the major psychoses in twelve general occupational categories ranging from unskilled to professional.

It almost goes without saying that his data, method, and findings raise many questions that should be considered before attempting some theoretical interpretation. The finding that the low prestige occupations have the highest rates of first admissions for the major mental disorders is—all things considered—hardly startling. However, without even touching on the difficulties here Frumkin barges joyously ahead and using the "method of *Verstehen* rather than by strictly empirical methods" proceeds to all kinds of generalizations about man in American society which are not only dubious in general but also in relation to his findings which should be boxed in by many qualifications. To illustrate with a few choice quotations:

> A man's occupation, in general, is more imporant to his mental health than is the occupation of a woman to her mental health.
>
> Thus, we find that the more radical male and female unskilled and service workers have the highest rates of alcoholic and syphilitic psychoses because, next to criminal acts, sexual promiscuity and alcoholism seem to be the best emotional outlets, the best known escape from hostility, the rejection and general frustation which are so often encountered among people in the lower socio-economic strata of our society.
>
> To go a step further, one might say that the etiology of lower socio-economic-status-group mental illness (and crime) is generally socio-genic in nature, whereas in the upper strata of society, mental illness is generally more psycho-genic. Thus, the etiology of middle class mental disorders, being somewhere in between, i.e., more or less equally socio-genic and psycho-

genic, or simply psycho-genic in origin, reflects characteristics of both extreme strata of society (13b).

I have used Frumkin's study here not because he reported his findings, but because his study illustrates well the trap of excessive sociological zeal as one moves from rate differentials to some theoretical interpretation. His quality of interpretation is too strained, but hardly drops as the gentle rain from heaven.

Turning now to the general theories that have directed both types of studies, I have already indicated that they are of the most broad and general kind. The epidemiological studies, particularly the European, have stemmed from a broad biological basis that attempts to point up differences or similarities which must be inferred about the genetic composition of a given population. Investigators with this orientation are likely to be more intrigued if the rates in a given population group or between different population groups are approximately equal.

The ecological studies, largely American, by contrast are likely to be guided by some broad sociological theory which attempts to show how certain social factors may be psychotic-inducing for persons occupying a given position in time or social space in a social system. From this broad theoretical basis excitement is high when a distribution of cases in relation to some aspects of social structure shows that the rates vary significantly from the different positions in that structure. The question, then, immediately arises: How certain can we be that our findings actually depict a "true" rate difference in the incidence of the disease or diseases under study? This is the rub, without doubt, for the fact is that with respect to both types of studies controversy has centered around the problem of validity. In general, those investigators with a bio-organic orientation try to show that significant rate differences of incidence in a social structure are not valid and cannot be accepted, while investigators with more of a sociological orientation attempt to show that they do possess a validity. Then there are, of course, the

stolid empiricists who attempt to examine the evidence with an impartial and objective eye.

I wish now to comment briefly on the purpose and value of these studies for an understanding of mental health and disease. It seems to me that these studies, considered jointly, have five central purposes and can be valued to the extent that they prove useful as aids to detection, diagnosis, treatment, and prevention of mental disorders in human society. First, these studies frequently serve the purpose of administration and can be helpful in pointing to the quantity and quality of services and facilities that may be needed in the future. Secondly, they may serve the purpose of sharpening our devices for detecting and screening the mentally ill in any population group. Thirdly, they can provide us with some conception of the size and extent of mental disease in general, or of a specific disease as it may exist in a given population group. Fourthly, these studies can add to our knowledge of social systems by showing those elements of processes within such systems that are associated with high and low rates of mental disturbances. And finally, they can sometimes be suggestive of hypotheses bearing on the etiology of mental disease that must be investigated by other methods and that must serve as a testing ground for the crucial relevance between biological and social psychological theories.

With these rather lengthy introductory remarks, I return now to my central task which is to critically examine the hypotheses utilized to explain findings of selected epidemiological and ecological studies of mental disease. This task has three parts. First, I wish to analyze briefly the central problem with which this type of research is always confronted. Secondly, I want to point to some of the characteristics of our data that present much difficulty for this survey-type of research in mental illness, especially in the complex, technologically advanced societies of the West. And finally, I intend to examine the hypotheses that have been utilized to explain rate-similarities or differences in various types of social structures. I further intend to examine these hypotheses as they have evolved in the

numerous studies that purport to find the incidence and prevalence of mental disease in different positions of the social structure or in specific delimited population groups. However, I do not intend here to cover the total literature, which is voluminous, but to confine my citations and illustrations to what, in my judgment are some of the most significant studies that have appeared during the last two decades.

The central problem which these studies present is posed by the question: "How does one define or delimit the case?" Now, this has been examined in other contexts (30) and I do not wish to belabor it here. But the problem continues to be crucial and is generally brought up when the results of such studies are presented. Currently, there are three devices: (1) There is the arbitary definition. Thus, we will count all persons who seek help from psychiatrists, clinics, or hospitals for the first time, all persons entering a mental hospital for the first time, or all persons being treated at a given time. However, if we arbitrarily define the case, how do we know if coverage was complete enough to assert the reliability of the rates as found so that any interpretations of them will have some validity?

(2) One can argue, as I have on occasion, that who is counted as sick in any social milieu is the result of a social judgment made by family members, friends, or neighbors and a judgment that the person must accept in some fashion by taking action or having some action taken upon him by others. This means that judgments about who is mentally disturbed or can be regarded as a mental case will vary in different social milieus, communities, and sub-cultures. This definition stacks the cards and contains within it a built-in explanation for rate variations at the different positions in social structure. This position also implies that the way to reduce mental disturbance in a high rate group would be merely to conduct some kind of educational campaign to bring about greater acceptance and toleration of human frailties. If this position has any relevance it probably would be to the minor type of disturbances, but this then becomes a clinical problem. How does the diagnostician deter-

mine that he deals with a minor personality distortion and one that is not a forerunner of a more severe disturbance?

(3) Of course, there has always been the hope that for those types of mental illnesses where etiology is still obscure, biochemical research will eventually find the answer and in so doing will perfect a test so that we can say with complete assurance that a given person has a given mental disease. Some clinicans have felt that these statistical-survey-type studies are useless without such an objective test. However, one might say in their defense that while they have contributed little of etiological significance, they have helped to clarify certain issues in the mental health field, to be revealing of our social systems and to point up certain hypotheses that might be investigated by other methods.

In addition to the problem of defining the case, there are several other characteristics of our data that make the conducting of this type of research quite difficult, especially with respect to our confidence concerning reliability and validity. Here, I have reference to the following factors: the great mobility of patients that is a reflection of stepped-up mobility in the entire society during the past half-century; the discrepancy between the onset of mental disease and the decision to seek treatment; the great spread of age at which patients are first recorded as having a mental illness, and the great variety of diagnostic types with numerous remissions in the various categories over the years. In addition, there is also the great difference in policies and administrative practices of states and countries that make it difficult to secure comparable data. Finally there are always the great variety of theoretical orientations that exist among psychiatrists as they attempt to arrive at a diagnostic judgment.

Now, the central issue in all these studies is whether there are reliable and valid rate differences for mental disease, or for a specific mental disease, between different positions in a social structure or a sub-culture. Consequently, in the remainder of this paper I will be concerned with examining the hypotheses

that have been advanced to explain rate variations among the different positions within a social system and also to show the manner in which various studies have tended to classify them or advance them as interpretations of their own findings.[2] Thus, I am not concerned in this analysis with pointing to the various correlational findings of mental disease rates and different indices for status, isolation, or population characteristic that have appeared in the literature. For the function of these correlations is merely to add support to rate differentials that a particular investigator has found present in a particular social structure. That schizophrenic rates are high in low income occupations and in occupations of low prestige as Clark has reported (4) or that the rate of mental disease in certain subregions of Texas shows a correlation with the number of psychiatrists in private practice of .40 as Jaco has reported (21) will not concern us here. For such correlations, quite numerous in the literature, while interesting, have added nothing with respect to throwing any light on the social etiology of mental disease or a specific mental disease.

The crucial problems concerning rate differentials are three in number. (1) With what confidence can we assert the reliability and validity of rate differentials or no rate differentials for mental disease as found in specific social structures? (2) What is the most plausible interpretation of the presence or absense of such rate differentials? (3) Does a particular interpretation provide us with some suggestive hypotheses about social causation that we can subject to some crucial test?

There are two classes of hypothetical interpretations concerned with providing adequate explanations for rate differentials of mental disease in social structures. These are (1) the

[2] While in accordance with my assignment, I have confined my analysis to those studies dealing with position in the social system, I am constrained to point out that the same problem arises when making cross-cultural comparisons. For example, consider the following questions: Does a given culture have more mental disease than another culture? Does a given culture have a higher incidence of one type of mental disease than does another culture? The problem as to whether all cultures have basically the same kinds of mental disease is relevant to a cultural analysis but is not pertinent to our concern.

non-theoretical hypotheses and (2) the theoretical hypotheses. By non-theoretical hypotheses I refer to those explanations that stem from no acceptable body of scientific theory but find their *raison d'etre* in some defect of method or some obstacle in the environment. Theoretical hypotheses refer to propositions that derive from or can be derived from some acceptable body of scientific theory. Within the class of theoretical hypotheses, we wish to distinguish three sub-classes—those on biological, social psychological and social system levels.

Let us turn to a consideration of four non-theoretical hypotheses. All of these have been suggested at one time or another for the purpose of showing rate differentials that have been reported as incidence rates are in reality not "true" incidence rates. Perhaps the oldest and perhaps the fairest argument has been the notion of "incomplete coverage." From this point of view it is contended that a given investigator has not included in his count all the new cases occurring in an area mainly because he cannot get to them. Thus, if he counts only first admission to mental hospitals, his count is off because he does not include admissions to outpatient clinics and private psychiatrists. If he includes these his count is off because he does not take account of those in the community who are sick but who do not get into treatment. This argument has been faced by numerous investigators in their studies in one way or another including Faris and Dunham, (12a), Goldhamer and Marshall, (15a), and Hollingshead and Redlich (19a). This, of course, is the demand for a "true" incidence figure, and until it can be secured many are likely to remain skeptical of any significant rate differentials that are reported.

In the nineteenth century the one readily acceptable non-theoretical hypothesis was the "law of distance." This "law" merely asserted that the rate of first admissions to a hospital varied inversely with the distance from the hospital. This is equivalent to a contemporary non-theoretical hypothesis that views variations in the first admission rates by geographical or status positions as merely a reflection in the differential avail-

ability of psychiatric facilities, whether hospitals or beds. This was one of Kramer's (23) conclusions when he studied the admission data from 1916–1950 at the Warren State Hospital in Pennsylvania. Ødegaard (34) reports that rates of admission vary in the different areas of Norway because the more and better facilities tend to account for the higher rates. However, in another study of the incidence of mental disease during World War II, Ødegaard (35) concludes that loss of facilities cannot explain the 1941–1943 decrease in rates. He notes that decreases were particularly marked in Oslo where facilities were fairly good and least marked in the northwest where the bed shortage was especially bad. He also notes that if facilities determined the decrease then there should be a decrease in nursing cases and readmissions but in these series there was an increase. He finally concludes that even though a number of psychoses seemed directly caused by the War, there was a real decrease in psychiatric morbidity and a net gain in mental health. This finding will concern us further when we attempt to assess those theories on the social system level.

Malzberg's (29a) evidence on this point for New York State is also negative. He shows that between 1925 and 1935 bed capacity increased 71.4 per cent while first admissions only increased 58.3 per cent. He thinks that this fact combined with evidence from other states seriously questions the proposition that bed capacity determines the rate of first admissions. Even so, as Ødegaard has pointed out to me an increase of bed capacity at a given point in time is, as a rule, followed by a jump in first admissions the following year.

There is, finally, the statistical criticism. While this stems from an acceptable body of statistical theory, I have called it a non-theoretical hypothesis because it disposes of rate differentials by claiming some defect in the collecting of or handling of the data. Thus, this hypothesis points to inadequate sampling, failure to establish significant rate differences, and inadequate number of cases—thus, by increasing the cases by three or four in any cell the entire picture might change—or a mobile popu-

lation. Any of these items might destroy any rate difference reported.

I turn now to a consideration of our first set of theoretical hypotheses: those concerned with a biological level of explanation. In general, these studies have been largely of European origin and have been directed to (1) establishing valid estimates of the "true" incidence and prevalence of mental disease and specific mental diseases in various populations, (2) collecting statistical evidence for the genetic linkages of mental diseases through specific family groups and/or inbreeding of populations (1b) and (3) obtaining reliable frequency figures for a population in order to compare them with frequency found in hereditary tainted families.

I do not intend to review all of these European studies but will point to two of them which illustrate the above points. Torsten Sjögren (41a) reported on the investigation of the occurrence of psychoses and oligophrenia during the period January 1, 1900, to December 31, 1944, on an island, A:bo, off the west coast of Sweden. He states, "The object of the investigation was a thorough statistical and hereditary-biological analysis of psychoses and oligophrenia as well as genealogical survey of the cases concerned and their facilities as far back as it was possible with the aid of parish registers and archives. . . . Furthermore, statistical investigations regarding heredity and incidence of mental disorders in the parents and siblings of the probands can be made and the extent of inbreeding analyzed" (41b). His data, after diligently searching the records, included 397 persons of which 335 comprised his proband group and 62 were secondary cases (siblings of probands). Of the total, 397 persons, 158 comprising 117 families were assembled into a connective pedigree complex. His findings broken down by pedigree, parish, birth, and residence, and diagnosis are voluminous, and while impressive, Sjögren presents them with no interpretation other than implied genetic explanations. His overall finding showed a prevalence rate of 11.0 per 1,000 population for psychoses and 5.7 per 1,000 for oligophrenia. He finds

this in excess of figures for all of Sweden in 1940 which were reported as 4.5 and 2.9 per 1,000, respectively. These latter figures he thinks, are under-enumerated. On the other hand, he finds that his figures are quite close to Stromgren's figures for the island of Bornholm in 1938 reported as 11.4 and 4.2 per 1,000, respectively. This comparison is reminiscent of that reported by Goldhamer and Marshall (15b) when they call attention to the fact that Kurt Fremming's expectancy for figures for various causes of mental disorders up to the age of 56 in the same island of Bornholm (1951) are of the same order and magnitude as those expectancy measures that Goldhamer and Marshall calculated for New York State.

In view of my distinction between the epidemiological and ecological studies of mental cases, it is of some interest to note the contrast between the distribution of Sjögren's cases in his pedigree complex as compared with those cases outside the pedigree complex in four parishes of A:bo. While Sjögren does not present these figures, a slight computation based on the population of the parishes which he has given us, reveals it. My computed rates on Sjögren's figures are shown in the accompanying table.

One notes interestingly enough that the cases in the pedigree complex are more highly concentrated in two of the parishes that are poorest in terms of social economic level while the cases outside the pedigree complex are distributed rather evenly over the four parishes although the higher rates are in the other two

Number and prevalence rates of cases of psychoses and psychopathy in four Parishes of A:bo in and outside of pedigree complex.

Parish	Average Population 1900–1944	No. in Pedigree Complex	Rate[1]	No. Outside Pedigree Complex	Rate[1]	Total Cases	Rate[1]
S	4,071	89	21.9	71	17.4	160	39.3
V	1,749	33	18.9	28	16.0	61	34.9
R	2,334	7	3.0	49	21.0	56	24.0
K	1,232	1	0.8	30	24.4	31	25.2

[1] Rate per 1,000 general population.

parishes. I am pointing this out because it seems contrary to what might be expected. Here, the expectation would be that the cases inside the pedigree complex showing hereditary linkages should be distributed more evenly over the four parishes, while the cases that supposedly have no genetic linkage should be more heavily concentrated in the two poorest parishes. This may, however, mean that the sick people on this Swedish island who are sick because of some genetic characteristic find themselves in much poorer circumstances than those who are sick for other reasons. Thus, those who are sick for other reasons come largely and somewhat evenly from all the social classes in this particular Swedish island.

Bremer's study (1a) of the frequency of psychiatric morbidity in a small fishing village in Northern Norway from January 9, 1939, to April 1, 1944, attempts to combine the genetic emphasis of the European investigators with the mental hygiene-environmental emphasis which he thinks is characteristic of some of the American studies. While making his observations his role was that of the local medical officer for the village during World War II. His findings, like Sjögren's, are quite detailed in breakdowns. Moreover, his figures are quite comparable as he reports an overall percentage of about 12.9 for psychoses and psychopathy combined and 5.56 for oligophrenia out of a total population of 1,080 persons over the age of 10 years. The figure would be reduced slightly if 245 children under 10 were included. He further goes on to examine the frequencies of the several types of psychiatric morbidity by occupation, race, migration, and wartime conditions.

His environmental emphasis is seen when he divides the total population into two groups, the secure group defined as self-supporting, 636 persons; and the insecure group described as on relief or spasmodically employed, 689 persons. He finds that the frequency of the psychoses are the same for both groups, the neuroses are more frequent in the "secure" group and psychopathy and oligophrenia are more frequent in the insecure group.

In these two studies there is a definite attempt to present evidence showing genetic linkage although there is a marked caution in interpreting the evidence. The other striking feature is the close agreement of the overall frequency for psychiatric morbidity. In truth, if the fact that no frequency variations for the psychoses and oligophrenia in the different kinds of communities in the Western world could definitely be validated, then there would be every reason to suspect that any variation of these disturbances within the social structures of a given Western society are spurious and could not be explained by social psychological factors or processes.

Several hypotheses on a social psychological level have been developed. These hypotheses tend to accept rate differentials by geographical areas and/or social space and emphasize social factors that are supposed to be causative to a given mental disease or a group of diseases. Thus, if the validity of any one of these hypotheses could be established and it could be shown how the factor operates then one would have a most satisfactory interpretation of any rate differentials by time, geography or social space. Thus, the "social isolation" hypothesis, first proposed by Faris (11) and later developed in our joint work (12j) was most applicable to schizophrenia.

Since publication of MENTAL DISORDERS IN URBAN AREAS there have been numerous references to the "isolation hypothesis" in the literature but only three studies have appeared which provide certain data for appraisal. Lemert (27) attempts to give support to this hypothesis, as we did, by showing the correlation between first admission rates of mental disease and the percentage of various nationalities by counties in Michigan. His work added nothing and could hardly be taken as any test of the isolation hypothesis.

Jaco (20) in a somewhat more ingenious fashion attempts to develop a kind of index of social isolation by interviewing with a prepared schedule a carefully drawn sample of residents in four census tracts in Austin, Texas. Two of these census tracts had high and low schizophrenic first admission rates and

the other two represented high and low manic-depressive first admission rates. He proposes nineteen null hypotheses to point up the differences in isolation between high and low schizophrenic census tracts. He rejects thirteen of these hypotheses and thus by this device infers that there is more social isolation in the high rate schizophrenic tract than in the low rate schizophrenic tract. In other words, he finds more schizophrenia in an area where persons show less contact and communication with one another. He, of course, does not show that persons who break down with schizophrenia are more isolated than those that do not.

This, of course, is just the point where Kohn and Clausen (22) begin their study, for they see clearly that if there is anything to the isolation hypothesis, it will have to be established through careful study of persons who develop schizophrenia as over against those who do not. They selected a sample of 45 schizophrenic and 13 manic-depressive first admissions to mental hospitals from Hagerstown, Maryland. They then secured a group of controls paired individually with the patients on the basis of age, sex, and occupation. The persons comprising the four samples were then interviewed through a schedule that covered the following topics: residential and occupational history, parental family relationships, friendships and activities of early adolescence, dating patterns, adult social participation, and a brief psychosomatic inventory.

They proceed to make careful qualitative analyses of their interview data and come up with the following findings: (1) About one-third of schizophrenic and manic-depressive patients, as compared to none of the controls, show evidence of being socially isolated at age 13–14; (2) there was no evidence that isolated patients were prevented from interacting with their peers because of lack of playmates, excessive morbidity, severe illness, or parental restrictions; and (3) no difference was found between patients and controls in their perceptions of family relationships.

The investigators recognize that their data are based on

retrospective impressions of a group of patients but find also that their data secured from 26 out of 30 patients check favorably with the material in the hospital records. Their general conclusion is that "the data do not support the hypothesis that social isolation in adolescence is a predisposing factor in either schizophrenia or manic-depressive psychosis." They think rather that in those cases showing social isolation that this is an indication that a person's interpersonal difficulties are so great that he cannot very well continue to function in this area. Rather, it is a question of how he got that way in the first place so that he takes isolation as a way out. Further research along this line is very much needed utilizing other research designs for the study of isolation in the early years.

Gruenberg (16) in his preliminary report on an ecological study of the old age psychoses in Syracuse, New York, raises the question as to whether the patients who were not living alone were experiencing a process of social isolation and that effort should be directed to discovering if such isolation is symptomatic or causative.

Another hypothesis on the social psychological level has centered around the issue involved in the migration of peoples. The central questions are: Do persons who migrate from one place to another have a higher rate of mental disease than persons who live out their lives in a given community? If they do, is this higher rate caused by persons who are prone to a given mental disease moving around or is the higher rate caused by the fact that migratory persons are subjected to more severe stress than persons that have stayed home? These questions have been thought at times to be particularly relevant to schizophrenia. During the latter part of the nineteenth century much effort was expended by commissions and analysts to show that the higher rate of mental disease among foreign-born was due to the defective character of the biological stock represented by the immigrants. This position was very much undercut by Malzberg's work (29b) when he attempted to analyze first admission data by age, sex, nativity, race, and economic group.

His results cast much doubt on the earlier position and at least serve to refute the notion that European countries were dumping their defective stocks on American shores. However, Malzberg's efforts cannot be taken as a disproof of the genetic factor in certain types of mental disease or for that matter a proof that rate differentials between native and foreign-born are the result of a difference in environmental conditions to which the different nationality groups have been subjected. Even so, it became somewhat fashionable in the 1930's to regard the higher rates of foreign-born and native-born of mixed parentage as due to the difficulties of adjustment to new cultural conditions.

Ødegaard (33) challenged this conception by his study of the Norwegians who had migrated to Minnesota. He interprets the higher rate of schizophrenic disorders among Norwegians who migrated to Minnesota as compared to Norwegians who stayed at home, as due to the fact that those who are more organically predisposed are most likely to migrate. But, like Malzberg, Ødegaard's evidence for the validity of his interpretation of rate differentials is inconclusive.

Tietze, Lemkau, and Cooper (44) in their study add no new dimension to the problem when they show that higher rates of psychopathy are found among those persons who move frequently as compared to those who reside for a long period in the same house. They found also that the rates were higher for intra-city migrants than for migrants from other communities. Again, one has difficulty in determining whether persons who migrate are more likely to be psychiatrically ill, or more likely to become psychiatrically ill because they migrate. One might develop a cultural integration hypothesis with regard to the above data. Persons who are firmly rooted and integrated in the culture of a community have a minimum probability of developing a functional mental disorder as compared to those who are not so well integrated.

Another hypothesis on this level is one that emphasizes the etiological role of a multiplicity of stress conditions in the social environment. This is a broad general hypothesis, less specific

than the two discussed above, and stems in a large degree from the work of Faris and myself (12a). Here, our work was set in a broad theory of social organization-disorganization. We portrayed the city as having certain areas of marked disorganization characterized by cultural conflict, minimum consensus, slum dwellings, high population density, and high land values. The idea was that these areas of social disorganization produced disorganized persons and consequently more crime, delinquency, sickness, mental disease, and suicide was to be expected in these areas.

Leighton's Stirling County Study has been particularly designed to deal with the multiple environmental stress hypothesis. While the final report of this study is not yet available, there have been several accounts in the literature indicative of the trend that the research has taken. Leighton (25) himself has provided a statement of theory with some derived hypothesis that the research will supposedly test. Dohrenwend, a social analyst on the project, has provided a rather complete statement of the theory and objectives of the study. In line with the attempt to get at the etiological significance of certain socio-cultural factors, Dohrenwend states the central hypothesis, "that social disorganization impinges on such needs of the individual as those for physical security, sexual satisfaction, the expression and securing of love, the securing of recognition and the expression of creativity, thereby producing psychological stress and disruption" (7). Again in line with their general objectives, Dorothea C. Leighton (26) reports on the prevalence of psychiatric symptoms in a small town of 3,000 population on the basis of record searches and interviews. She reports that a much wider distribution of psychiatric symptoms exists than is commonly believed and estimates that approximately 37 per cent of the adult population in this town are psychiatric cases whether under treatment or not. This bears a close relationship to the study by Rennie and Srole (38) where they attempt to show the prevalence of certain psychomatic conditions in relation to social class.

These last three hypotheses derive rather clearly from a type of social psychological theory. The hypotheses, broadly considered, can be viewed as inferences from differential rate distributions of mental disease, particularly schizophrenia. I turn now to a series of hypotheses which derive from what I have designated as the social system level. In this sense society is viewed as a functioning social system through space and time that sifts and sorts certain vulnerable personalities so that they get into (a) environments where the probability of a mental breakdown is increased or decreased, or (b) certain sub-cultural pockets that serve either to precipitate or induce a mental breakdown. The general hypotheses here can be designated as "social selection." In other words, certain persons because of age, sex, personality traits, intelligence, emotional instability, psychotic proneness, are selected for certain positions in occupational groups, city areas, marital status categories, institutions and the like in contrast to other positions in these structures as the social system moves through time. This process may be either active or passive as far as the person is concerned and through it one can account for significant differences in the rates for mental disease.

There are approximately four hypotheses that appear in the literature that represent various ways of stating the more general hypothesis of social selection. These four hypotheses are:

1. That certain persons because of personality inadequacies or mental disease proneness have a tendency to drift into certain social classes, sub-cultures or city areas.
2. That visibility of, and tolerance for, mental disorder vary with the attitudinal structure of different types of communities.
3. That certain persons because of their psychic needs to break their social ties tend to select and segregate themselves in areas, cultural or spatial, marked by anonymity.
4. That as the size of the city decreases rate differentials between socio-economic areas tend to disappear.

My intention at this point is to examine several of the more

significant epidemiological studies in this area which have appeared during the last fifteen years and to show the manner in which these studies have called upon these hypotheses for negation or validity of the rate differentials that they have reported. In the earlier Chicago study by Faris and myself our procedure was primarily empirical even though set in the social organization-disorganization theoretical framework. We merely started out with the question as to whether or not the distribution of mental disorder would follow the pattern of rates that seemed to characterize the distribution of other social problems in the city (12b).

The contemporary research worker in this area, being more design conscious, generally sets up a series of statistical hypotheses which he purports to test for validity. This procedure is well-illustrated by Jaco's study of the distribution of mental disease in Texas (20) and the Hollingshead-Redlich study of the prevalence of treated mental disease in the class structure of New Haven (19a)[3].

Thus, Jaco begins his study with three hypotheses:

1. The probability of acquiring a psychosis is not random or equal among subgroups of the population.
2. Inhabitants of different areas exhibit different incidences of psychoses.
3. Persons with different social attributes or affiliations have different incidence of psychoses.

Jaco finds that his evidence gives support to his three central hypotheses. He is well aware that there may be a significant

[3] This study will, in all probability, become a classic for showing the influence of the class factor on mental disease rates. There are numerous aspects of this study that deserve comment, but our concern is with attempt to explain the rate variations by social class. The authors themselves recognize that social-cultural factors affect the prevalence of treated disorders in the population but do not represent essential or necessary evaluations in the etiology of mental disorders (p. 360). Dr. Redlich himself, to a question along this line, stated: "The New Haven study has not really brought out anything of etiological significance in explaining prevalence and prevalence itself is not a very good measure from an epidemiological viewpoint." (43). Further, it is of some interest that S. M. Miller and E. G. Mishler in their expository review of this volume, decide not to discuss the issues raised concerning the relationship of social factors to the etiology of mental disease, but rather to consider the study's implications for psychiatric practice. (31).

gap between "true" and "treated case" incidence. However, his findings might as easily be interpreted through the general social selection hypothesis as they can be with respect to "industrialization," "anomie," and "enculturation." The interesting fact that his findings in certain instances run counter to the findings of other studies should give one pause. For example, his high rates for both males and females among professionals and semi-professionals cannot be easily explained away by the "marginal status" supposedly enjoyed by this group in Texas. Again, his low rates among Spanish-Americans for both males and females are unexpected and contrast rather sharply with the high rates frequently reported for various ethnic groups in northern states. This might be caused by the fact, as Jaco suggests, that the Spanish-Americans are not well-integrated into the dominant Anglo-American group and also have a very protective kinship system. It may also be explained by the hypothesis that the visibility and tolerance for mental disorders in this group is at a variance with the dominant Anglo-American group. Again, much more research will be needed to show that a closed, intact, integrated group has less mental disorder than one that is less integrated around a common core of values. Eaton's findings for the Hutterites might prove to be an example of a negative case.

Now, the point I wish to make is that when Jaco's findings run counter to findings of other studies, like other investigators he tries to account for them by social factors even though the total evidence is contradictory. His hypotheses are broad statistical statements, which, while no doubt true enough in terms of his evidence, provide us with no basis for accounting for the rate differentials as found in age, sex, marital, occupational, or ethnic groups. He could just as easily have used some hypothesis centering on "social selection" which he seems fairly close to doing when trying to interpret the lower rates among the different age and sex categories of Spanish-Americans.

Let us examine now the Hollingshead-Redlich hypotheses, also three in number:

1. The prevalence of treated mental illness is related significantly to an individual's position in the class structure.
2. The types of diagnosed psychiatric disorders are connected significantly to the class structure.
3. The kind of psychiatric treatment administered by psychiatrists is associated with the patient's position in the class structure.

Much the same sort of criticism can be centered on this study of social class and mental illness. While their organized data give support to their hypotheses, their study is largely of prevalence and our concern must perforce be with incidence rates, for these should give some clues as to whether social factors bear some relationship to the etiology of mental disease. Here the expectancy would be that if social class factors have some relevance for etiology one would expect that incidence rates for a given disorder would have a significantly inverse relation to the class structure. Further, one would have to establish that the higher rate of persons in the lowest class is made up of persons who originated in that class. This is under the assumption that the conditions of life at a lower class level in our society are more likely to be psychotic-inducing than other kinds of life conditions enjoyed by other social classes.

Now, Hollingshead and Redlich do have several tables of incidence rates constructed from those persons who entered treatment for the first time during their six months observation period. When they compute the rates based on these cases for the four social classes, they find, using the chi-square test, that the difference is significant at the 5 per cent level. However, the rates do not vary inversely with class as the following table[4] shows:

Class	*Rate*
I–II	97
III	114
IV	89
V	139

[4] Source: Text Table p. 212, Social Class and Mental Illness.

These data are, of course, based on all the cases of mental illness that entered treatment for the first time. When this total group is broken up into neurotic and psychotic groups, the incidence rates for the neurotics do not vary with class structure. Here, Class IV has the lowest rate and Class III the highest rate with Classes I–II and V having rates of 69 and 66, respectively. However, in the case of the psychotic group the incidence rates vary with the class structure but the differential in the first three classes is probably not significant while Class V rate is twice that of the other rates. This might indicate that Class V contains many more psychotics than might be expected provided that they started in Class V. This the authors later try to show in discussing the drift hypothesis. Their Table 17 shows that schizophrenic incidence rates vary with the class structure although there may be questions of whether or not the differences between the classes are significant or the case basis for the classes is sufficient for claiming a difference.

In this study these data, as presented above, might constitute the evidence for arguing that the influence of life conditions on a given class level plays some role in the development of a particular psychosis. But the case is not proven beyond any doubt. The concentration of the higher incidence of psychoses in Class V might just as easily be regarded as due to some form of social selection—a hypothesis which is not stated in their work. The denial that the neuroses are linked to class (19b) does not quite stand up as the prevalence rates for neuroses vary directly with the class structure and is certainly a function of the fact that the upper social levels in New Haven society have more money to spend on this type of personality perturbation.

In one of their articles, Hollingshead and Redlich suggest a social selection hypothesis when they state that "current prevalence is a measure of the responses patients in the several classes make to the treatment process." This, they label as the "differential treatment hypothesis" (18).

Some data from England serve as a challenge to the New Haven study. In an address before the British Sociological So-

ciety, Dr. J. N. Morris (32) presented the following rates for England and Wales.

First admissions for schizophrenia (per 100,000 men age 20 and over) in 1949–53 in the five social classes.

Class	I	II	III	IV	V
Rate	51	57	93	103	229

He then asks the ever-present question "Is this fact caused by a downward drift of men to Class v or by the social and familial environment in Class v that tends to produce an excess of schizophrenia?" He then points to a study that has classified a national sample of schizophrenics, ages 25–34, by occupation and social class and then classified their fathers by occupation and social class on the basis of an examination of birth certificates. The answer was clear. There was an excess of schizophrenics in Class v but their fathers were distributed rather evenly over the five classes. This seems to indicate that the patients experienced a downward drop because of their illness and to explain their excess in Class v. Thus, here in this study, the schizophrenic illness operates as a selective factor or, from the opposite angle, the social system functions to place these men in Class v.

Lilli Stein's study of class and schizophrenia (42) is relevant here because her findings also contradict some American results. She proceeds by selecting four East London boroughs (E) outstanding for their high proportion of men in Classes IV and V and five West London boroughs (W) outstanding because of their high proportion of men in Classes I and II. She then tabulates the first admissions to mental hospitals from E and W boroughs for 1954–1955 by age, sex, and three diagnostic groups—schizophrenic, manic-depressive and psychoneurotic. The results run contrary to those found for American cities. Her findings showed that the W boroughs had significantly higher rates in practically all age and sex categories as compared to the E boroughs. This held true, in general, for schizo-

phrenia and psychoneuroses; it was less marked in the manic-depressive group. She then proceeds to distribute these data among the social classes in the two sets of boroughs. While the rates are still higher in W she does find in both areas that they vary inversely and consistently with the class structure and for all three diagnostic groups. While she concluded that there was a real class gradient in the inception of schizophrenia she thinks these results must be qualified because of the radical differences between the E and W boroughs in the sex ratio, number of persons living alone, types of private households and origins of the population. These differences signify that a single index of social class may not be too meaningful.

In another study from England, Carstairs and Brown (3) attempt to get at the incidence and prevalence of psychiatric disorders in two different types of communities, Rhondda, a coal mining region, and Vale, an agricultural area. Rhondda had a more densely concentrated population than Vale which was rural and spread over a wide area. Rhondda was found to have more psychiatric cases than Vale (3.8 to 2.6 per 1,000 population). They also found that in Rhondda psychiatric cases were more heavily concentrated in the non-miner group. This might be regarded as an instance of the vulnerability of a minority group but the investigators are more inclined to think it is an example of adverse selection. They conclude that the linking of social pressures to psychiatric disorders must wait upon an analysis of the social structure and value system of a community, the circumstances leading to the emergence of declared cases and the carrying of undeclared cases in each community and the clinical features of the illnesses.

Ødegaard has been one of the most constant proponents of the social selection hypothesis. From his earlier study of emigration (33) where he emphasized the tendency of the psychiatrically vulnerable persons to migrate to his more recent analysis of psychiatric cases in relation to the occupational structure of Norway, (36) he has generally attempted to show that social selection versus environmental stress provides the

most enlightening explanation for the rate differentials in various social structures. His study of the incidence of psychoses in the various occupations analyzes 34,457 first admissions to Norwegian mental hospitals, 1926–1950. In general, he finds the highest admission rates among those occupations with the lowest social prestige. He finds the highest rate in the seamen of the merchant marine. This holds for all psychotic groups with the exception of epilepsy, mental deficiency, and manic-depressive psychosis, although in these diagnostic categories the seamen are second from the top rate. He explains this by a social selection process as well as a secondary selection which takes place when men around 30 seek other occupations leaving the unstable and psychopathic types in the seaman's group. His finding that the high rates of manic-depressives are in the more favored occupational groups is in conformity with American results, but the mechanism of social selection is more evident for schizophrenia and for the psychoses with epilepsy and mental deficiency. He also thinks that social stress and protection are not explanatory of the distribution of psychoses in the occupational structure.

It is of some significance to note that the hypothesis of social selection is frequently urged when dealing with such special structured institutions as the army, merchant marine, or prison. Winston in her study of psychoses in the army (45) asks "Does mental disease act as a further selective factor in the already highly selected military group?" While she reports no tendency for mental disease to increase in the army, she does find that the psychological misfits are weeded out eventually so that mental disease tends to decrease as length of service increases. This was the opposite of Ødegaard's findings with respect to the merchant seamen but probably means only that the seamen make a voluntary choice to leave the service while the army actively weeds out those who cannot fit into the rigors of army life.

Sims (40), in a study of noncommissioned officers in the British Army, Dominion Army, and prisoners-of-war, also sug-

gests that the difference in the incidence of the disease in the various groups is due to high selection standards. He further reports that there is, in addition, a secondary selection as many recruits are weeded out in basic training and this lowers the incidence among those who get overseas. Ekblad (10) also supports Ødegaard's social selection hypothesis when he shows that his sample of seamen have higher rates for psychopathy and schizophrenia when compared with non-seamen in naval training.

Now, I have pointed to these studies to illustrate the application of the social selection hypotheses when applied to certain rate differentials in selected social structures. It seems to be much clearer in application when dealing with occupational, marital status, and specific institutional structures than it is when applied to social classes or geographical areas. In the latter, the problem is much more complex although even here the social selection hypothesis must be considered. In essence it is an hypothesis that explains significant rate variations as due to the manner in which a given social system functions through time and in its functioning tends to sort and sift persons into class and community positions.

As we indicated above there are a number of more specific hypotheses that stem from this general hypothesis and these I now wish to examine. Both the "drifting" and "segregation" hypotheses are variations of social selection. "Drifting" implies an involuntary segregation and results from the automatic functioning of a social system through time. "Segregation" implies a voluntary, conscious selection of a preferred place of residence in a community. The "drifting" hypothesis came into focus over twenty years ago as an attack on the rate distributions for the various psychoses in Chicago and Providence that Faris and I (12a) presented in our joint work. It was quite a theme for some reviewers at the time for it questioned the significance of our rate patterns. At the time we argued that the concentration of the younger catatonics and paranoids were quite similar to the concentration of older cases and this fact argued against

the thesis that the pattern could be explained by "drift" alone. This issue was examined by investigators by locating the addresses of a sample of schizophrenics from Buffalo twenty years prior to their first admission to state mental hospitals during 1949–1951 (24). They concluded that the concentration of schizophrenic cases in low-income areas was not the result of downward drift from better areas. They further showed that the high rates are not caused by the drift of men living alone.

Hollingshead and Redlich (19a) in their study also faced this issue since much of their results would be dependent upon the fact that mental patients are not excessively mobile by class. They examined for their schizophrenic group four sets of data —the nativity of schizophrenics compared with the nativity of adult population in the community; the birthplace of native-born schizophrenics in relation to class; the past addresses of schizophrenics, and the class position of their families of orientation as compared with their own social class position. From these data they concluded that there was no evidence of a downward class drift of schizophrenics. In fact, the evidence showed that 91 per cent of the patients were in the same class as their families of orientation. These data raise certain questions about the class mobility of patients. What is the class mobility of the residents of the community itself? Are schizophrenic patients less mobile by social class than non-schizophrenics in the community? One might well expect that they would be but how then does one account for those observations that schizophrenics are more likely to be geographically mobile than non-schizophrenics.

Prior to the Buffalo study, Gerard and Houston (14) on the basis of studying an ecological distribution of male schizophrenics in Worcester, Massachusetts, concluded that the high rates of schizophrenia in the low social-economic areas are to be largely explained by patients living alone. They found that patients coming from families showed marked residential stability and those patients not in families showed marked residential instability. They suggest the hypothesis that these non-family

men used residential instability as a means of protecting themselves against involvement in disruptive family relationships. Thus, they imply the segregation hypothesis.

Hare (17) uses this hypothesis to explain the concentration of schizophrenic cases in Bristol, England. He starts out by trying to check on our findings for Chicago to determine if there is additional evidence for supporting the social isolation hypothesis. He shows that, like our findings, the schizophrenics were concentrated in the central areas while the manic-depressives were more widely distributed. He points out that the high rate in both "good" and "poor" central areas is related to the factor of living alone—thus supporting Gerard and Houston. He also raises the alternative, namely, that the concentration of schizophrenics is to be explained either by segregation or the causal effect of the environment.

Another hypothesis that is a variation on the social selection theme stresses the differential visibility and tolerance of areal populations for mental abnormality which, in turn, accounts for rate differentials in different communities. Mary Bess Owen (37) first raised this question years ago when suggesting that this hypothesis might explain our contrasting distributions of paranoid and catatonic schizophrenics. In a more recent study (2) there was an attempt to test the hypothesis that if a difference in demand for hospitalization causes higher urban rates, the urban excess should be predominantly in the rate for cases whose symptoms are sufficiently tolerable socially that care outside of a hospital would be possible. They then proceeded to examine the rural and urban first admissions in Western Ontario, broken down so as to give rates for severity of symptoms for age groups 45–64 and 65 and over. Their results showed that for the age groups studied the excess urban admission rate is almost entirely explained by the greater urban tendency to hospitalize cases whose symptoms would be tolerated in a rural community. They conclude that this device may be useful in distinguishing between the real and apparent difference in rates of mental disease.

Finally, there is the hypothesis that as the size of the city decreases the rates tend to approach a parity for the different socio-economic areas. This proposition was demonstrated rather sharply by a study (5) of the distribution of first admissions of schizophrenics in Hagerstown, Maryland. The findings are extremely pertinent for they contradict the Chicago findings and by implication raise questions about the spurious quality of the rate differences found in a large city. The findings show that there is no difference between the schizophrenic rates in Hagerstown and the remainder of the county and no difference between the rates in five areas of Hagerstown arranged on the basis of a rental value index. They also show that the absence of areal rate differences cannot be explained by shifts in diagnosis or unusual upward or downward mobility. Five possible interpretations of these findings are offered but the one favored by the investigators is that the differences in socio-economic areas of a small city, like Hagerstown, are not sharp enough to show the differences that the large cities produce. They conclude with the question: "In the constellation of values, attitudes, behaviors, and relationships that are generally indexed by socio-economic status, by occupation, education and area of residence, what are the factors that are crucially related to schizophrenia?"

In this review of social structures and mental disorders, I have attempted to concentrate on the various hypotheses that purport to explain significant rate differentials in selected social structures. The fact that the rate distribution in different social structures varies for the different diagnostic groups makes the attempt to interpret total mental disease rate differentials as of doubtful etiological value. From an environmental perspective, interest is greater for the so-called functional disorders but even here the evidence is highly inconclusive for asserting with any confidence that a high rate in a given position of a social structure is a product of certain stresses, strains, and conflicts in that position.

I have also attempted to show that certain basic methodologi-

cal orientations about the nature of man in human society enter into the way one views these rate differentials. This is partially reflected in the contrast between the European epidemiological studies and the American ecological studies of mental disease, and between those investigators with a biological orientation and those with a sociological orientation.

Biological, social psychological, and social system theory have been productive of various hypotheses to account for significant rate differentials in social structure. Biological hypotheses have largely been interested in the comparative count of psychiatric cases in different population groups. Here, rate differentials are to be explained away. In contrast, social psychological hypotheses are likely to assume that rate differentials have been reliably established and then to validate the hypotheses by other research designs. To date, the results on this level have been inconclusive.

On the social system level, the hypotheses have been largely directed to showing how the rate differentials are functions of the manner in which the social process works in communities and social structures. Here, one has certain sociological hypotheses that give us added knowledge about the social system but are not stated in a form that can show the relevance of socio-cultural factors for the production of specific mental abnormalities. The task which the social scientists face here is to be able to show that certain stresses from a given position in a social structure make such an ingression into a given human experience that his mental content and behavior emerge in such bizarre forms that he cannot be fitted into the social structure at any point. Then, too, from the standpoint of prevention there is a need to know the positions in the social structure to which the more bizarre behavior types can become adapted. Probably, it is not without point to note that man in addition to trying to correct certain behaviors and make them more acceptable, must also learn to live with the type of personality structure that, through events and experiences, he has made for himself. In any event, the task of the social scientist in this

area is not likely to be facilitated until the clinician has developed methods for isolating those persons who will not break down with a mental disease no matter how extreme the adverse, traumatic, or discouraging elements in his life will be. On the other side of the ledger, however, it may be that these epidemiological studies of specific mental diseases may provide some clues for distinguishing between different kinds of abnormal personalities that are caught up in the network of a present diagnostic category.

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DISCUSSION

DR. DUNHAM: I thought I might indicate some of the assumptions that I had in mind in writing this paper. I hope that it has agitated the rest of you as it has Professor Jaco. We were having a discussion about it. It is a great subject of controversy.

What I had in mind, of course, is the strikingly contradictory interpretations of various ecological and epidemiological studies dealing with mental disease. While there are contradictory findings, there are many more contradictory interpretations of these data.

I want to say a word about my assumptions. I am assuming that schizophrenia eventually will be more reliably diagnosed. What con-

stitutes schizophrenia now obviously covers a multiplicity of reaction types. We want to separate out these types—I mean some sort of a core group as over against other types now called schizophrenia.

The second point I would like to make repeats one that was made earlier this morning about the social values in the community—those significant values surrounding the patient or person who goes to a treatment facility and which determines entrance into the facility. While this may be true, it says nothing about the different degrees of pathology, or disorganization, or disturbance, which these patients may have.

It seems to me this is fundamental, because when we talk about these values we are really talking about the differential tolerance of different community settings. While this is all right to talk about, it is something separate from the pathology of the individual patient.

The third point is that I am taking for granted that epidemiological studies should go on. If we develop some more ingenious designs, we may be able to throw some light on differences in schizophrenic groups and, also, we may be able to get some leads in etiology which might be investigated by other methods.

Dr. Ernest M. Gruenberg: Because of a last minute cancellation by the invited discussant Dr. Morton L. Levin, it has fallen to me to prepare some introductory remarks to start the discussion. I found great difficulty in disagreeing with Dr. Dunham's paper but by applying myself thoroughly to the task I have managed to find a few points which I think need to be made.

The first concerns the two definitions of ecology and epidemiology, where ecology is defined as the study of the environment and life history of the organism in contrast to epidemiology, which is called the sum of what is known about epidemics. I don't think this is a very useful distinction, for hardly any existing epidemiologist would define epidemiology so narrowly.

Although not all would accept as broad a definition as John Gordon gives, "epidemiology is medical ecology," I think many of us would accept the notion that this is a fair approximation of the correct definition. This, then, would make epidemiology a special field of your ecology. But since the purpose of getting together is to review our state of knowledge regarding the causal factors which affects the occurrence of mental disorders, it is not really necessary, I would say,

that we agree with one another as to which is the most appropriate word usage; by your definition we are all ecologists here.

It is clear also, I believe, that those who are preoccupied with the physical features of the environment or with the gene characteristics of the organism, are well aware of the importance of the social and cultural environment and, contrariwise, those preoccupied with the social environment appreciate the importance and relevance of the physical environment.

As far as I know, no one in this room is an advocate of a brainless psychiatry, or of a mindless psychiatry, or of an asocial psychiatry.

Toward the end of your classification of the uses and difficulties of doing these studies, Dr. Dunham, you raise the possibility of getting out of some of the difficulties by considering, as cases of mental disorders, persons who are assigned this characteristic by the social environment, using what some people call the social definition of mental disorders.

I really don't think you mean to say that this concept of a social definition or identification of case is equivalent to the cases who, in fact, come to clinical attention as patients of psychiatrists, even though I got an implication from the text that this meant that there was no other mode of socially identifying a person as mentally disordered.

Further on, you discuss the use of biochemical identifiers, or an analogue of biochemical identifiers, as a way of getting around the problem of case selection for research.

Here again, I felt that I couldn't go along with you all the way as it was written, because I don't see how such an identifier gets us out of the problem that we are confronted with. For any such identifier must be validated. First, the identifier has to be found, and to do that you have to discover what proportion of your cases give positive results, and what proportion of those giving positive results are real and not false positives. To do this you have to have an independent set of criteria regarding the nature of the case.

I don't see how such identifiers would save our souls in any way. For it seems to me that there is no general answer to the question, and any particular study is quite justified in selecting the clinical entity it wishes to investigate. The suitability of the criteria used for identifying cases depends, first, on the relevance to the entity to be studied and, second, on whether or not the criteria used introduces

bias in locating and identifying cases in the sub-samples of the population which they intend to compare.

For example, taking the very simple classification by social classes, will the criteria give a higher proportion of the cases existing in the population in one social class than in another social class? Is the method of case-finding and the criteria applied biased with respect to the variables that they are going to be analyzed by?

In the discussion of Ødegaard's and Malzberg's data regarding the availability of beds in hospitals as related to the variation in mental hospital admission rates, I got the impression that you used their data and their arguments against their idea that mental hospital admission rates might be a function of bed availability. Both Ødegaard and Malzberg give examples where the increases in rates that they are pointing to do not coincide with a greater number of beds per capita in the population at risk. However, it seems to me that the concept of availability of beds is much broader than the number of beds per capita existing within a jurisdiction; surely there are other meanings of availability, some of which you mentioned earlier. I couldn't see why you were so easily persuaded by this measure, for it seemed to go against some of your earlier arguments.

In reference to the Hagerstown studies by Clausen and his associates on social isolation, I wondered if you would comment more on two features of those studies. One of the things that is peculiar about their findings is that there wasn't any difference in the manic-depressive cases and in the schizophrenic cases with regard to the social isolation found in youth. I would be interested to hear your discussion on the theoretical implications of the failure to find differences.

The second part of this study[1] (which I agree is of great importance to the field you are discussing), although it found no differences in social environment factors of the youths who later became cases, they did find that the cases had had less social interaction. Would not this in itself, at the age of 13 or 14, regardless of the causes, have later effects which would fit into some of the theoretical concepts that you have advocated in the past? Could not this fact be regarded as a causal factor even if the cause of their failure to take opportunity for social interaction was not clearly understood?

[1] Kohn, Melvin L.; Clausen, John A.: Social Isolation and Schizophrenia. *American Sociological Review*, 1955, 20: 265–273.

The last point I will bring up at this stage is that while you raised a number of models for explaining the concentration of cases of schizophrenia in various sections of the population—usually in the center of cities—and while you described various features of the different drift processes, there was one model that has been suggested which I didn't find, but perhaps in my haste I might have missed it. This is the notion that most people who spend the early part of their lives in the center of our cities move out of them by adult or early adult life, and that the central concentration of cases is due to their failure to migrate away from the center rather than because of their migration to the city's center. Such a model proposes that in a socially mobile society the standard pattern is mobility upward and, therefore, the concentration of cases occurs as a residue rather than as drifting into lower social groups. It seems to me that the analysis of the data within the framework of such a model would give us slightly different results.

The last point that I wish to discuss (and which isn't original with me), is that upward mobility may, in fact, be a protector against the development of some of these syndromes; that this relationship accounts for the high concentrations of cases which are found in an environment unfavorable for personality development which increases the risk of developing schizophrenic psychosis. This kind of hypothesis, it seems to me, would be very easy to test.

The easy way to test it would be to take a random sample of people living under undesirable conditions and split them in half, giving one-half of them a lot of help in moving upward socially and comparing their experience over the next ten years with those to whom one did not give a lot of help.

Finally, I thought I would like to mention one administrative implication which seems to me quite clear. As with so many other health problems, much of the mental disorder data which Dr. Dunham reviewed shows a high concentration of pathology in that part of the population which has the least financial resources to pay for help and for preventive activities.

This relationship once again, as it has done so many times previously, raises the question as to why our physical and mental health services should be operated on the basis of the ability of lower income groups to finance these services, when, in fact, we know that those who can produce the most in the way of taxation have the least need

for the services, while those who have the least ability to raise the tax money have the most need for services. In other words, the whole doctrine of the development of health services out of local resources seems to me to be incompatible with the kind of data that has been presented here.

In closing, I would like to thank Dr. Dunham for a very interesting and stimulating paper.

SUMMARY OF DISCUSSION

1. Movement in an urban environment, particularly at the present time was classified into two types: the first being intentional movement, the second being forced.

Individuals or families might elect to change residence because they had moved up in the social scale or for some other reason. Forced movement, on the other hand, could occur under such circumstances as urban renewal, where families had to leave neighborhoods where their roots might have extended back for years, even generations. A study of both these types of movement might throw light on some of the points raised earlier on stresses, on how different types of people in each of the two groups handled their stresses, and what the consequences were.

2. Issue was taken with Dr. Dunham's view that epidemiological studies of mental disorders support theories of genetic origins when the statistical differences are at a minimum.

It is well established that, in many diseases where a genetic factor has been demonstrated, considerable rate differences between populations occur. These differences result from the several factors that influence gene frequencies in populations, such as migration, genetic drift, and selective fertility.

Genetic explanations, where rate differences between population are small, as in schizophrenia, suggest that whatever genes are involved are rather widely distributed and, therefore, not as subject to drift, migration, etc. However, one instance investigated by Dr. Böök showed a 3 per cent rate in a population in the north of Sweden as against a 1 per cent rate in a population in the south. This appreciable difference, based on carefully checked diagnosis and apparently representing a true variation, cannot be taken as evidence against genetic etiology. They can be regarded as suggesting that the

living conditions and culture in the north favor survival of people with the relevant genetic makeup more than do conditions in the south.

3. Further discussion on the implications of findings regarding differences in the incidence of a given disorder, such as schizophrenia, in different populations and different cultures, emphasized the importance of the investigator's basic approach to these data. One approach seeks out constancy in incidence rates and thereby justifies fatalism regarding preventive work, while the other, which looks for variations in these rates feels it can justify a belief that preventive manipulations might bring high rates down to the lower levels of the low rates. Concomitantly with this line of reasoning, is the tendency to suggest that constancy of rates implies a biological origin of an illness while variation in rates a social origin. Strong issue was taken with one or both of these inferences. However, no disagreement was expressed over the statement that, since these assumptions did affect so many people, findings emphasizing variation or constancy did tend to affect practice.

4. Dr. Dunham in his paper suggested that many were skeptical of rate differentials of mental illnesses which depended on first admission rates to mental hospitals. Dr. Kramer, however, emphasized that variations in first admission rates were well established but that disagreement arose over their interpretation. "True" incidence must account for both first admissions hospitalized during the index year plus the new cases which developed during that year but which were not hospitalized. It was this last factor—the new cases which were not hospitalized—which created difficulties.

Dr. Kramer suggested that the problem be examined in the following manner. If:

Iy = "True" incidence rate for index year (y).
Cy = All cases developed during year (y).
Ry = First admission rate for year (y).
Ay = Cases developed and first admitted during year (y).
Ax = Cases developed prior to year (y) but first admitted during year.
Az = Cases developed during year (y) but not admitted during year.
P = Population at risk during year (y).

$$Iy = \frac{CY}{P} = \frac{Ay + Az}{P}$$

Then:

$$(I). \quad Ry = \frac{Ay + Ax}{P}$$

In formula (I), first admissions are separated into those cases who developed during the index year (Ay), and those who developed any time before the index year (Ax). Now, if the latter group (Ax) were to be replaced by the group of cases which developed during the index year but were not hospitalized during that year (Az), the numerator would become the total of cases who developed during the index year (Cy); in other words, this is the numerator needed for finding the "true" incidence rate. In effect, the first admission rate departs from the "true" incidence rate by substituting cases who were admitted during the index year but who developed prior to that year (Ax), for those cases who developed during that year but who were not admitted during this period (Az).

Dr. Kramer then performed the following manipulation on formula (I):

$$Ry = \left(\frac{Cy}{Cy}\right) \cdot \left(\frac{Ay + Ax}{P}\right)$$

(multiplying the rate by a fraction equal to unity does not alter the rate)

Producing the following formula:

$$(II). \quad Ry = \left(\frac{Cy}{P}\right) \cdot \left(\frac{Ay + Ax}{Cy}\right)$$

Since the "true" incidence rate for the index year (Iy) equals the total number of cases developed during the year (Cy) divided by the population at risk (P), we may substitute, Iy for $\frac{Cy}{P}$

$$(III). \quad Ry = (Iy) \cdot \left(\frac{Ay + Ax}{Cy}\right)$$

Thus it is evident that the first admission rate (Ry) is a function of the "true" incidence rate (Iy) multiplied by a factor which relates the total number of newly developed cases (Cy) to the number of cases who were new admissions (Ay) and the number of old cases

who were first admissions (Ax). This factor can be called the coefficient of the "true" incidence rate in the first admission rate; or, more simply, "the coefficient."

But in comparing two different situations the difference between the two coefficients might be large enough to severely distort, or even reverse, the rates being compared. Was there, Dr. Kramer asked, really a higher incidence of schizophrenia amongst single men than among married persons as admission rates seemed to indicate? Or was this merely due to the protective influence of marriage which was more likely to keep a case at home rather than to throw him into the hospital?

While in all the published works of Malzberg or Ødegaard various explanations were given for observed difference in admission rates, no data were offered to support the validity of the difference with respect to true incidence.

In all epidemiologic studies that dealt with hospitalized populations, it was considered important that true incidence and first admissions be related. Otherwise the interpretation of the observed rate differentials had to remain dubious.

5. This view, in turn, was attacked in the belief that a true incidence rate could never be attained. Dr. Dunham's paper considered that the social definitions of who ought to be hospitalized affected the rate at which the people were hospitalized, implying that it was necessary to penetrate the effects of these varying definitions in order to get at the picture of true incidence. But what was considered to be a suitable case for hospitalization, and how people considered disease, affected the course of the disease. From this point of view the problem was not one of weighing the relative effects of pathology on the one hand, and of social tolerance on the other, but rather of appraising the effects of social attitudes on the development of pathological reaction patterns.

If this were the case, then the "true" incidence rate, in the terms of the previous discussion, was, in fact, unattainable.

DR. DUNHAM: I want to thank the discussants for their critical remarks, and I think I will start at the middle and work around to Dr. Gruenberg, because I think Point 3 hits at what is a crucial aspect of the formulation.

It was said that if you take the position that there is a uniformity of incidence among the various peoples throughout the world, then this fact would discourage the search for methods of prevention. I am not so sure whether I would agree with that, although I do think that if you could establish that there are marked differences in incidence of mental illnesses among peoples throughout the world, this would encourage a search for sociological explanations of mental disease, as over against biological formulations.

On the other hand, I agree that these things are not completely mutually exclusive because the human biological organism always interacts in an environment and culture.

Even if one accepts this, it seems to me that there would still be various therapeutic possibilities, even though a given mental disease might be distributed fairly evenly among the populations of the world.

The very last point, Point 5, is very intriguing to me because in some of my own papers I have argued in the same way: that how people conceive of a person's behavior or syndrome, and so forth, will affect the process or the course of the disturbance. In fact, one might argue that this is in some ways an operational definition and that if we could really measure these judgments then we would not have to look further: this *is* the disease. If I have shifted my position, it is probably because of various kinds of influences and new evidences that have been brought to my attention.

On the other hand, as I said in my opening remarks about my assumptions, that while I agree that this may be true, at the same time there may be a biochemical basis for certain of these diseases, but you could still have the course of the disease being affected by the attitudes and values that are prevalent in the community.

We probably will come back to that again. I don't think that the issue is settled by any manner of means, since it represents two ways of looking at the same phenomenon.

I think that I agree with everything that Dr. Kramer remarked about the interpretation of hospital admission rates (Point 4), though I will have to examine his formula more carefully. I think that his comment on skepticism over the significance of rate differentials is borne out by the whole paper, for I am skeptical of the interpretations too, regardless of whether we can get true incidence or not. The whole paper is concerned with this matter of evaluation and interpretation, particularly contrasting those of the social scien-

tists with those of the medically-trained people. It strikes me in reading the literature that the medical person in his type of study has always been able to say that other factors account for the difference as found; while if you move to the Hollingshead and Redlich type of study, they are trying to show that social class factors in some way affect the incidence and prevalence of the behavior.

Moving on to Point 2—the discussion about the relation of genetic theory to unvarying incidence rates—the interpretations are the same as mine, if I understood them correctly. I was saying that if there are no statistical differences or if they are at a minimum, this would imply an acceptance of the biological position and in this I did not take account, (and it is quite true) of the possibility of gene drift or of the selective factors in reference to genes in the population. I have sometimes argued—and Dr. Böök is the authority here, and I am not—that in genetics you ought to have a random distribution of the disease throughout the population whereby if there was something to the role of social factors, or stress factors, or cultural conditions as affecting the incidence of mental disease, then the disease would not be randomly distributed throughout the population.

I agree with the statement about spontaneous and forced migration (Point 1). It certainly would be interesting to make a comparison between those who move because they want to improve their situation, as over against those who are forced to move. Also it seems to me there is a third category—those that seemingly just drift or never get started any place. This was somewhat the burden of Dr. Morris' interpretation in England, and his findings, which contradict those of Hollingshead and Redlich, have already been referred to. One interpretation was that the fathers, who are evenly distributed by social class, had sons who never got off the ground, so to speak. These sons were counted in the lower class position because they never seemed to have the push, or the energy, or whatever it may be, to do something to change their situation.

To come to Dr. Gruenberg's opening remarks. First, we might speak of biochemical identification. I am not a biochemist, of course, but I am informed from various sources that there are supposed to be advances on this front. However, when we examine the evidence it often seems to go up in smoke and we have had many disappointments. At the Lafayette Clinic they seem to think they are close to a biochemical breakthrough. I don't know.

Dr. Gottlieb, head of the Clinic, sometimes tells me "If we can perfect some biochemical identifier, why, we will have a more objective designation of these cases, and we will have a sounder basis for an epidemiological study."

I don't know whether any biochemical test will ever be perfected or not, but it has struck me—and I speak primarily of schizophrenia —that if this should happen, it would certainly seem to me—and I know it has been the dream of the people in this area—that you would have a device for saying "These cases respond positively to the test, but those do not." Those schizophrenics who do not respond to the test—those we have been calling schizophrenic—are probably adjustment problems. We would then have an opportunity of making comparisons between this group and the ones responding to the test, assuming other etiological factors or forces would be operating in the negative group. At least, that was the way I tended to look at the thing.

You asked me about the Hagerstown studies, referring particularly to the one by Clausen and Kohn on isolation in schizophrenia, where they found that one-third of the manic-depressives and one-third of the schizophrenics reported the same amount of isolation in their early adolescent years.

I think that was interesting, despite its defects as a retrospective study. It is a backward glance, of course, but Clausen and Kohn seem to try to show (and they spent a lot of time doing this) that in the situation of these patients there was no reason for them to be isolated in the sense that they did not have any playmates or as many people surrounding them as the persons that were used as controls. The situations of both study and control groups were very similar.

Therefore, when you raise the question as to why they were isolated, you will again have somebody here who will say that, whatever is the matter with the person, and whether or not he does go out and interacts, he will probably tend to withdraw and be by himself, consequently other people will leave him alone. To account for why the manic-depressives and schizophrenics are the same, I have no interpretation here. Probably there was something the matter with their diagnosis! I remember when I was doing a study years ago on catatonics and brought some of the recovered patients to be examined, the psychiatrist invariably changed the diagnosis and said they were manic-depressives.

Dr. Gruenberg also made a remark toward the end about the possibility of upward mobility: that this is one of the values prized in our particular type of society, and that it might be preventive of disturbance.

I was reminded of a theoretical paper that Warner gave 20 years ago when he argued the reverse: that striving upward may produce a kind of tension. This idea was quite popular during the 30's and as one looks back on a lot of that literature, one wonders how much it was frequently bound to the particular social, cultural, and psychological climate and situation of the period. Those interpretations really could not be supported by any hard and fast evidence; so that while one might make a proposition one would be able to test, it strikes me—to refer back to Morris' study—that many of these schizophrenics may not get started upward at all because of the nature of their disturbance.

Perhaps this will show that I have tried to respond at least to all of your comments, and that I appreciate your remarks.

CHAIRMAN LEIGHTON: Thank you, Dr. Dunham.

I will not try to summarize the meeting today. There have however been a number of general themes that seem to run through the various papers and the discussions that might be useful to point out; and I may also take this occasion to slip in some reactions that have been turning over in my mind.

One matter is the problem of recognizing the phenomenon with which we are concerned, psychiatric disorder. This involves the conceptual and methodological question of standardization, of establishing standards for comparison. This in turn has at least two main components: The first is the problem of pattern—what kind of pattern of human behavior are we going to be concerned with; and the other concerns the question of impairment—of the degree to which these patterns handicap the individual in the social medium of which he is a part.

A serious matter in this problem of standardizing our point of reference, is the question of environmental components that may be part of the diagnostic process itself, that is: the built-in environmental factors that are there because of the way diagnoses are made. Such can obviously lead to misconceptions of cause if not recognized.

Another related problem that has been touched upon today, is the

one that comes up when we ram together the total collection of what is seen in a mental hospital and call it mental illness—schizophrenia, psychoneurosis, alcoholism, sociopathic types of behavior, etc. This is certainly an exceedingly heterogeneous group of human behaviors, and it makes one wonder whether he would generalize about admissions—first admissions or any other—to a general hospital as is commonly done about first admissions to a mental hopital. Yet, after all, is a general hospital any more heterogeneous in its collection of different kinds of things than a mental hospital?

A third problem is the question of what are noxious factors: What are to be considered as damaging factors in the origin, the course and the outcome of the phenomena we call psychiatric disorder? There is a need here to give thought to benign and neutral aspects. An otherwise damaging constellation of events can be rendered neutral or even benign because of particular factors which enter. Hysterectomy is an example: in one situation it is stressful; in another situation it may be psychologically benign.

A point which I think is pretty closely related to the previous one, is the need for an explicit statement of the psychological frame of reference in thinking about cause. This has been implicit but only implicit all day in many of the things that have been said. Various assumptions were made as to how psychological mechanisms work and yet the underlying framework that any one of us holds could be quite different from others in the group here. So it would seem to me desirable to have these assumptions made more articulate than they were. It might be wise to have clear concepts as to the framework of ideas as is the case in other forms of epidemiology with regard to infection, nutritional deficiencies, etc. I think there was some obscurity in our implicit references as to how something might be benign or noxious so far as the origin, course and outcome of psychiatric disorders are concerned.

The last point has to do with the emphasis that was given to the necessity for interplay between the epidemiologic approach and the clinical and laboratory approach. The separation of these is an unhappy situation. Allied to this is the business of interrelating extensive study with intensive study. While we can very often get one kind of reliability, if not validity, with an extensive study which deals in large numbers, we are apt to lack knowledge as to why we get these associations. The intensive small study that is geared to the ex-

tensive study may provide at least something better than exists now.

Mr. Allen, the late editor of *Harper's,* used to tell a story which I think is apposite to the fix we are in here. He said he had a roommate at Harvard who didn't study very hard but lay around, apparently thinking. Along towards the third year Allen got out of this man what he was doing. He said he was spending his time trying to be famous, and that the quickest way was to have a law named after you: thus Newton was known for Newton's Law and so on. In his fourth year he hit upon it: "If you play with anything long enough, it will break."

That set Allen to work, and he established Allen's Law, which states that "Everything is more complicated than you think."

Before I turn in my gavel, I want to express my very deep appreciation to all of you for making the task of the chairman so easy today.

CAUSES OF DEATH RESPONSIBLE FOR RECENT INCREASES IN SEX MORTALITY DIFFERENTIALS IN THE UNITED STATES

PHILIP E. ENTERLINE[1,2]

FOR nearly every country in the West, the male excess in mortality has been rising persistently since about World War I.[3] In 1938, Wiehl noted this trend for the United States.[4] Subsequently, Bowerman recorded considerable increases in ratios of male to female death rates for the period 1900–1945, with the greatest increase in the age groups 15–24 and 45–64.[5] Sowder and Bond have called attention to a steady rise in the ratios of male to female death rates as each generation goes through life. This rise is becoming progressively steeper.[6]

It is the purpose of this paper to present recent data on trends in sex mortality differentials in the United States; to identify age groupings where the increases in sex mortality differentials have been greatest; and, in these age groupings, to determine which causes of death have been mainly responsible.

TRENDS IN THE UNITED STATES

Figure 1 presents annual age-adjusted death rates for white males and females for the period 1900 to 1958.[7] During most

[1] Division of Public Health Methods, United States Public Health Service.

[2] The writer is indebted to Nicholas E. Manos for helpful suggestions on this manuscript. Some of the material presented is from the writer's unpublished dissertation: A Study of Factors Associated with Male-Female Differentials in Mortality, The American University, Washington, D. C., 1960.

[3] Stolnitz, G. L.: A Century of International Mortality Trends, II. *Population Studies*, July, 1956, X, pp. 17–42.

[4] Wiehl, D. G.: Sex Differences in Mortality in the United States. Milbank Memorial Fund *Quarterly*, April, 1938, XVI, pp. 145–155.

[5] Bowerman, W. G.: Annuity Mortality. *Transactions of the Society of Actuaries*, June, 1950, II, pp. 76–102.

[6] Sowder, W. T. and Bond, J. O.: Problems Associated with the Increasing Ratio of Male over Female Mortality. *Journal of the American Geriatrics Society*, October, 1956, IV, pp. 956–962.

[7] Death rates have been age-adjusted by the direct method to the total 1940 United States population. For the period 1900 to 1933, death rates are for the expanding death-registration States. Death rates for United States nonwhites have followed much the same pattern as death rates for whites. To facilitate this presentation, death rates will be studied for whites only.

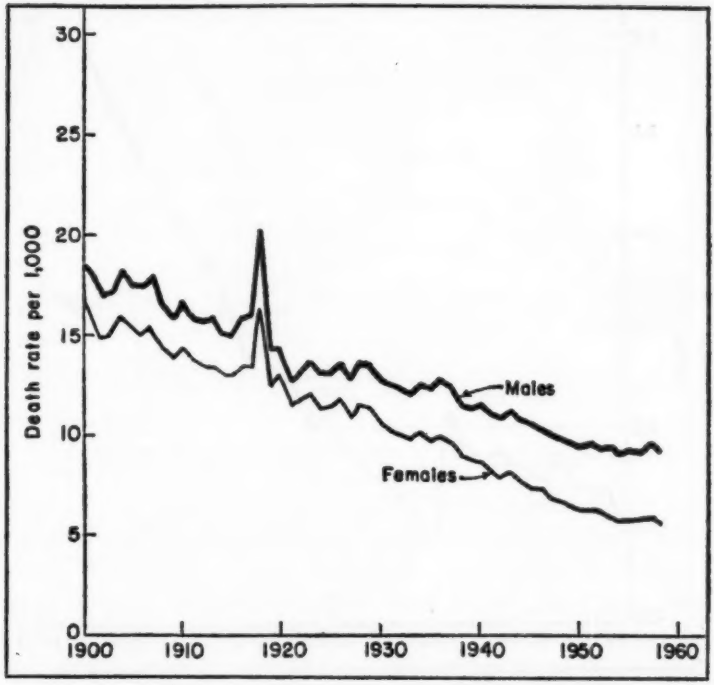

Fig. 1. Age-adjusted death rates per 1,000 population for white males and females, United States, 1900–1958.

of this period, death rates have declined for both sexes. The effect of the flu pandemic in 1918 is evident, causing a somewhat greater increase in the male than in the female death rate. Around 1920, death rates for males and females did not differ greatly. Starting in 1922, however, death rates started to diverge and have been diverging fairly steadily ever since.

Sex mortality differentials are customarily measured by the ratio of the male death rate to the female death rate.[8] This will

[8] This is sometimes expressed as the percentage the male death rate is of the female death rate. The choice of ratios of male to female death rates rather than absolute differences is arbitrary. Ratios of death rates remain unchanged only if death rates change by the same percentage. Where one death rate exceeds the other, if death rates decline by the same absolute amount ratios will increase. For a discussion relevant to this, see: Sheps, M. C.: An Examination of Some Methods of Comparing Several Rates or Proportions. *Biometrics*, March, 1959, xv, pp. 87–97.

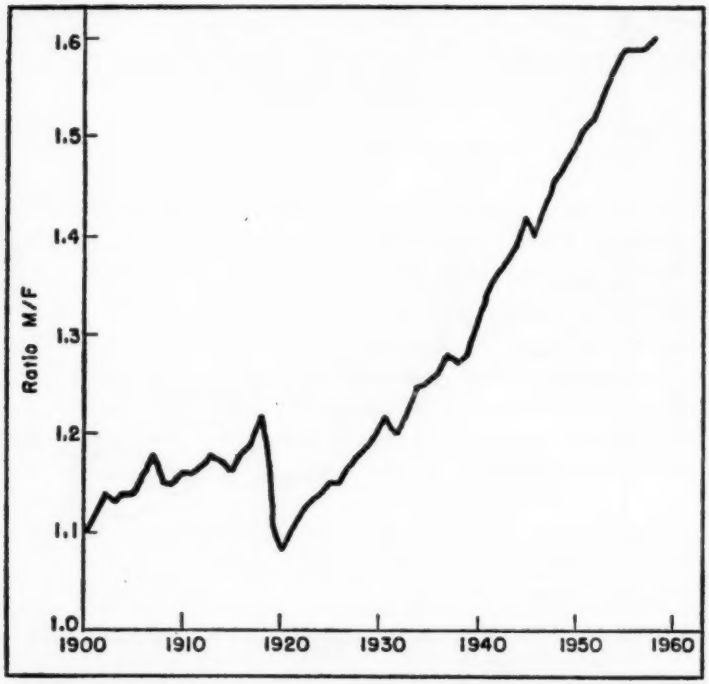

Fig. 2. Sex mortality ratio, age adjusted death rates, for white persons, United States, 1900–1958.

be referred to here as the sex mortality ratio and is shown in Figure 2 for the United States white population for the period 1900 to 1958. Three different linear trends appear: a trend for the period 1900 to 1918; a trend for the period 1920 to 1939; and a trend for the period 1940 to 1958. During the period 1920 to 1958 the ratio of white male to white female age-adjusted death rates increased from 1.08 to 1.60.

Trends in Selected Age Groupings

Figure 3 shows average annual sex mortality ratios by age for the United States white population for the years 1929–1931, 1939–1941, 1949–1951, and 1956–1958.[9] These ratios have in-

[9] Sex mortality ratios are based upon average annual death rates for the three-

(Continued on page 315)

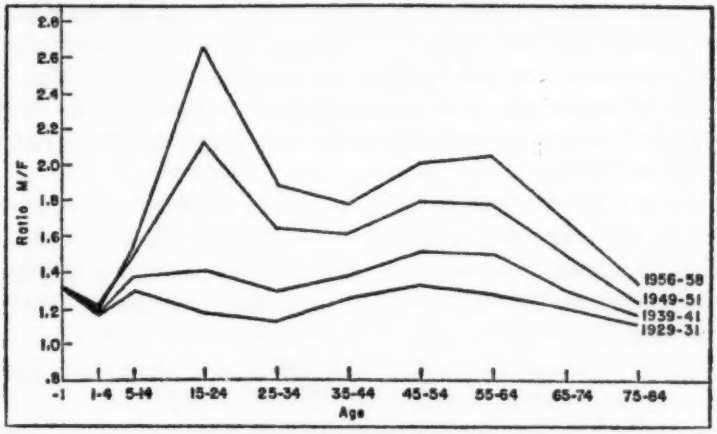

Fig. 3. Average annual sex mortality ratios for white persons for selected age groups, 1929–31, 1939–41, 1949–51, and 1956–58.

creased rather steadily at most ages. An exception is the age group 15–24, where a considerable jump occurred between 1939–1941 and 1949–1951. Since 1929–1931 the increase in the age group 15–24 has been the largest of those shown. In 1956–1958, the male death rate was over 2½ times the female death rate. Large increases also occurred in the age groups 45–54 and 55–64. In these age groups the 1956–1958 male death rate was twice the female death rate. There was relatively little change in the ratio of male to female death rates in the youngest and oldest age groups shown.

Contributions of Selected Causes of Death to Increases in Sex Mortality Ratios in the Age Group 15–24

During the period 1929–1931 to 1956–1958, the sex mortality ratio in the age group 15–24 increased from 1.164 to 2.646, an increase of 1.482. Table 1 shows the contribution of selected causes of death to this increase during three periods: 1929–1931 to 1939–1941; 1939–1941 to 1949–1951; and 1949–1951 to 1956–

year periods shown. Here and in the remainder of this presentation, sex mortality ratios will be studied starting in 1929. This largely eliminates any bias which might be introduced because of the expanding death-registration area.

1958, and shows the sum of these contributions for the period 1929–1931 to 1956–1958.[10] The death rates upon which these computations are based appear in Appendix Table A. Causes of death shown are those constituting 3 per cent or more of all white male or female deaths in the age group 15–24 in either 1930 or 1950.

Over a third (36.3 per cent) of the increase in the sex mortality ratio was due to trends in death rates for tuberculosis. This is because, in the past, tuberculosis was an important cause of death among young females; now, it causes relatively few deaths. In 1929–1931, over a quarter of all deaths among white females and 13 per cent of deaths among white males aged 15–24 were due to tuberculosis. In 1956–1958, only 0.9 per cent of white female deaths and 0.3 per cent of white male deaths in the age group 15–24 were due to tuberculosis.

In much the same manner, the decline in death rates from diseases associated with childbearing has affected the sex mortality ratio for all causes in the age group 15–24. In 1929–1931, deaths from deliveries and complications of pregnancy, childbirth, and the puerperium accounted for 15 per cent of all deaths among white females in the age group 15–24; whereas in 1956–1958, this cause of death accounted for less than 5 per cent of all deaths in this age group. As Table 1 shows, nearly a third (30.6 per cent) of the increase in the sex mortality ratio during the period 1929–1931 to 1956–1958 was due to the decline in maternal mortality.

[10] Estimates in Tables 1, 2, and 3 are based upon the derivative of the ratio of male to female death rates (calculus):

$$\Delta r = R\left[\frac{\Delta m_1}{M} - \frac{\Delta f_1}{F}\right] + R\left[\frac{\Delta m_2}{M} - \frac{\Delta f_2}{F}\right] + \ldots R\left[\frac{\Delta m_n}{M} - \frac{\Delta f_n}{F}\right]$$

where:

Δr = absolute change in ratio for all causes during period.
R = average ratio for all causes during period.
Δm = absolute change in male death rate for selected cause during period.
Δf = absolute change in female death rate for selected cause during period.
M = average male death rate for all causes during period.
F = average female death rate for all causes during period.

This formula is exact only for small time intervals. For the time intervals shown it is approximate, and contributions by individual causes do not add exactly to the total increment in the sex mortality ratio. Reasonable estimates of the relative contribution of selected causes are provided, however.

Motor vehicle accident deaths have also been a factor in increases in sex mortality ratios in the age group 15–24. Unlike tuberculosis and maternal mortality, however, this cause of death has become increasingly important. Its contribution to the increase in the all-cause sex mortality ratio is the result both of a general increase in death rates for motor vehicle accidents and an increase in the ratio of male to female death rates for motor vehicle accidents. Between 1929–1931 and 1956–1958 the white male death rate (per 100,000) in the age group 15–24 rose from 43.3 to 66.9 while the white female death rate rose from 12.5 to 15.2. The sex mortality ratio for motor vehicle accidents rose from 3.46 to 4.40 during this period.

Other causes of death shown on Table 1 did not contribute greatly to changes in the sex mortality ratio during the period 1929–1931 to 1956–1958. Deaths from malignant neoplasms, influenza and pneumonia, and suicide had virtually no effect while heart disease tended to increase and accidents, other than motor vehicle, to decrease the sex mortality ratio.

The "All other causes" grouping, shown in Table 1, is making an increasing contribution to the all-cause sex mortality ratio, accounting for nearly a quarter of the total increase during the period 1949–1951 to 1956–1958. An examination of this category reveals some contribution by many causes of death, with the largest coming from deaths due to rheumatic fever. Here, female death rates have declined more sharply than male death rates, with the result that the sex mortality ratio has increased.

In general, it would appear that the medical and public health advances which have caused declines in death rates from tuberculosis and maternal mortality have been an important factor in the increase in the sex mortality ratios in the age group 15–24. In view of the low death rates now achieved for these causes (see Appendix Table A), it seems unlikely that they will be of any great importance in determining future changes in sex mortality ratios. A factor which is likely to be important in the future is deaths from motor vehicle accidents. The general increase in deaths from this cause among our youth is probably a

Table 1. Contribution of selected causes of death to change in ratio of male to female death rates. White persons aged 15–24.

	1929–1931 to 1939–1941		1939–1941 to 1949–1951		1949–1951 to 1956–1958		1929–1931 to 1956–1958	
	Algebraic Contribution	Relative Contribution	Algebraic Contribution	Relative Contribution	Algebraic Contribution	Relative Contribution	Algebraic Contribution	Relative Contribution
Total Increment in Ratio	.274	100.0	.712	100.0	.492	100.0	1.482	100.0
Contribution Made By:								
Tuberculosis	.135	49.3	.230	32.3	.172	35.0	.537	36.3
Malignant Neoplasms	.002	.7	−.004	−.6	.017	3.5	.016	1.1
Heart Disease	.026	9.5	.049	6.9	.038	7.7	.112	7.6
Influenza and Pneumonia	−.011	−4.0	.025	3.5	−.020	−4.1	−.006	−.4
Maternal Mortality	.127	46.4	.222	31.2	.104	21.1	.453	30.6
Motor Vehicle Accidents	.024	8.8	.094	13.2	.051	10.4	.170	11.5
Other Accidents	−.072	−26.3	−.032	−4.5	−.003	−.6	−.106	−7.2
Suicide	.010	3.6	.008	1.1	.015	3.0	.033	2.2
All Other Causes	.021	7.7	.091	12.8	.116	23.6	.229	15.5

Table 2. Contribution of selected causes of death to change in ratio of male to female death rates. White persons aged 45–64.

	1929–1931 to 1939–1941		1939–1941 to 1949–1951		1949–1951 to 1956–1958		1929–1931 to 1956–1958	
	Algebraic Contribution	Relative Contribution	Algebraic Contribution	Relative Contribution	Algebraic Contribution	Relative Contribution	Algebraic Contribution	Relative Contribution
Total Increment in Ratio	.195	100.0	.299	100.0	.219	100.0	.713	100.0
Contribution Made By:								
Tuberculosis	.011	5.6	−.004	−1.3	−.023	−10.5	−.016	−2.2
Malignant Neoplasms	.029	14.9	.061	20.4	.068	31.1	.158	22.2
Diabetes	−.003	−1.5	.035	11.7	.016	7.3	.048	6.7
Strokes	.005	2.6	.016	5.4	.044	20.1	.065	9.0
Heart Disease	.121	62.1	.138	46.2	.086	39.3	.345	48.4
Chronic Nephritis	.018	9.2	.027	9.0	.007	3.2	.052	7.3
Influenza and Pneumonia	.005	2.6	.004	1.3	.001	.5	.010	1.5
Motor Vehicle Accidents	.003	1.5	−.012	−4.0	−.003	−1.4	−.012	−1.7
Other Accidents	−.017	−8.7	−.010	−3.3	−.008	−3.7	−.035	−4.9
Suicide	−.011	−5.6	−.005	−1.7	−.001	−.5	−.017	−2.4
All Other Causes	.032	16.4	.046	15.4	.032	14.6	.110	15.4

reflection of the increased availability of automobiles to them. It is not clear why young males have responded to this differently than females. Here would seem to be an important area for sociological investigation.

Contributions of Selected Causes of Death to Increases in Sex Mortality Ratios in the Age Group 45–64

In the age group 45–64 the sex mortality ratio for white persons increased from 1.297 in 1929–1931 to 2.010 in 1956–1958, an increase of .713. The contribution of selected causes of death to changes in the sex mortality ratio for this age group is shown in Table 2. The death rates upon which these computations are based appear in Appendix Table A. The causes of death shown are those making up 3 per cent or more of all deaths among white males or females aged 45–64 in either 1930 or 1950.

None of the causes of death contributing heavily to increases in the sex mortality ratio in the age group 15–24 are important contributors to increases in the age group 45–64. It would not be expected, of course, that diseases associated with childbearing would be an important cause of death in this age group. Many deaths among middle-aged persons are caused by tuberculosis and motor vehicle accidents, but Table 2 shows that these did not contribute to the increase in the sex mortality ratio.

Deaths from heart disease made, by a considerable margin, the greatest contribution, accounting for nearly half of the total increase during the period 1929–1931 to 1956–1958. Deaths from malignant neoplasms also contributed heavily, with the greatest relative contribution in the more recent periods. Contributions by heart disease and malignant neoplasms are due mainly to a general increase in death rates for these diseases (see Appendix Table A) and an increase in the ratio of male to female death rates for these diseases. Figure 4 shows that for

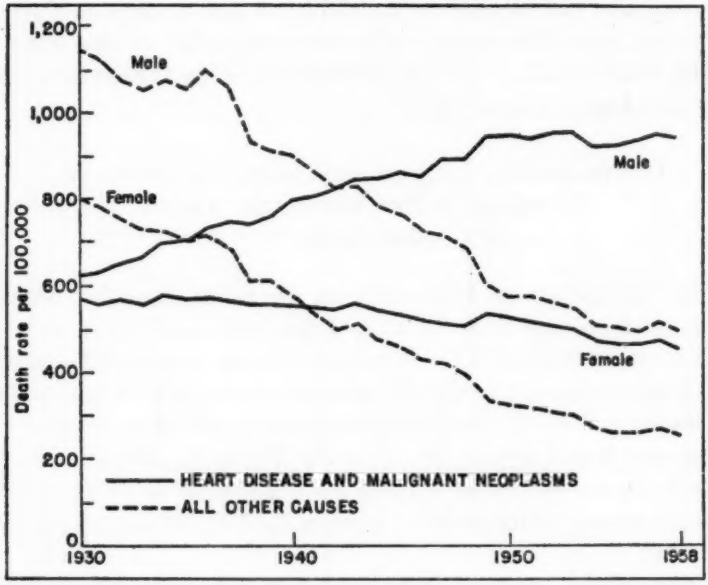

Fig. 4. Death rates per 100,000 population for heart disease and malignant neoplasms and for all other causes, by sex, white persons aged 45–64, United States, 1930–1958.

middle-aged white males death rates for heart disease and malignant neoplasms increased rather steadily during the period 1930–1958; whereas for females death rates for these diseases declined. For all other causes of death combined, the trends in male and female death rates were quite similar. Sex mortality ratios for heart disease and malignant neoplasms rose sharply during the period 1929–1931 to 1956–1958—from 1.50 to 2.99 for heart disease, and from .70 to 1.14 for malignant neoplasms.

Table 3 shows the contribution made by selected categories of heart disease and malignant neoplasms to increases in the sex mortality ratio during the period 1949–1951 to 1956–1958. The death rates upon which these computations are based appear in Appendix Table B. Data for previous periods are not shown because the manner in which deaths from the heart diseases were classified for statistical purposes prior to 1949 is

	Algebraic Contribution	Relative Contribution
Total Increment in Ratio	.2194	100.0
Contribution Made By:		
Cancer		
Mouth, Pharynx, and Esophagus	.0004	.2
Stomach	−.0007	−.3
Intestines and Rectum	.0075	3.4
Pancreas	.0016	.7
Lung	.0274	12.5
Prostate	−.0009	−.4
Kidney	.0010	.5
Bladder	−.0001	−.05
Breast	−.0025	−1.1
Uterus	.0256	11.7
Lymphoma	−.0003	−.1
Leukemia	.0003	.1
Other Cancer	.0089	4.1
Heart Disease		
Rheumatic	.00002	.01
Coronary	.0533	24.3
Myocardial Degeneration	.0026	1.2
Hypertensive	.0304	13.9
Other Heart Disease	−.0009	−.4

Table 3. Contribution of selected heart and neoplastic diseases to change in ratio of male to female death rates. White persons aged 45–64, 1949–1951 to 1956–1958.

not comparable to the method used starting in 1949, nor is it as satisfactory or meaningful.

Trends in four causes of death shown in Table 3 made large contributions to the increase in the sex mortality ratio. Trends in death rates for cancer of the lung and of the uterus were responsible for about a quarter of the increase; trends in death rates for coronary heart disease and hypertensive heart disease were responsible for another 38 per cent.

The contribution by cancer of the uterus was due to a decline in the white female death rate from 43.6 to 33.0 per 100,000. The contribution by hypertensive heart disease was due to a large decline in the death rate for this relatively low sex mortality ratio disease, coupled with an increase in the sex

mortality ratio—from 1.11 to 1.23. The contributions by lung cancer and coronary heart disease were due to increases in death rates among males—from 55.1 to 78.8 per 100,000 for lung cancer, and from 519.9 to 561.6 per 100,000 for coronary heart disease—while female death rates for these diseases changed only slightly.

Combining information shown on Tables 2 and 3 gives some clues as to factors responsible for increases in the sex mortality ratio among middle-aged persons during the past few years. Table 2 shows that trends in death rates for strokes made a contribution to the increase in the sex mortality ratio during the period 1949–1951 to 1956–1958. This was due to trends in death rates similar to those which have taken place for hypertensive heart disease. Death rates declined for both sexes, with the greater decline among females.

A major factor in strokes as well as hypertensive heart disease is high blood pressure. The decline in mortality from these diseases may well be the effect of the blood pressure lowering drugs introduced in this country around 1950. It is not clear why females should benefit more than males. Even if this were not so, however, a decline in mortality from strokes and hypertensive heart disease in excess of the decline from other causes would increase the sex mortality ratio for all causes of death combined. This is because of the relatively low sex mortality ratios observed for strokes and hypertensive heart disease and a decline in the weight applied by these diseases in the all-cause complex.

Discussion

The increase in sex mortality differentials has been attributed by some to biological rather than environmental factors.[11] It is generally believed that females have a greater constitutional resistance to degenerative diseases than men and that this is now coming to light as the result of declines in the infectious

[11] United Nations. Demographic Yearbook, 1957. New York, United Nations, 1957, p. 7.

diseases. This theory was proposed by Lenz to explain upward trends in sex mortality ratios for infants.[12] Later, it was extended by Herdan to all ages,[13] and more recently was presented by Madigan as the most likely explanation for increasing sex differentials in the United States. [14]

The data presented here lend little support to a biological explanation for the increase in the sex mortality ratio. Death rates for certain infectious conditions which have been unfavorable to the young adult female in the past have declined and this decline is responsible for much of the increase in the sex mortality ratio in the age group 15–24. It is not degenerative diseases which have emerged as the leading causes of death in this age group, however, but rather it has been deaths due to motor vehicle accidents. Traditionally, male accident death rates have been high, and a slight increase in this male excess combined with a decline in death rates for which a lower male excess or a female excess prevailed has been an important factor in increases in the sex mortality ratio among young adults. The male excess mortality from motor vehicle accidents is probably more a socio-environmental than a biological effect.

An important trend in death rates, particularly as it affects trends shown in Figure 2, is the trend in the age group 45–64. Death rates are high in this age group relative to the age group 15–24 and thus weigh more heavily in the all-age death rate.[15] Infectious diseases do not seem to be importantly involved in increases in the sex mortality ratio in the age group 45–64. Rather, trends in death rates for malignant neoplasms and the cardiovascular diseases are making a major contribution. While it seems reasonable to explain static sex differences in mortality

[12] Lenz, F.: Die Übersterblichkeit der Knaben im Lichte der Erblichkeitslehre. *Archiv fur Hygiene und Bakteriologi,* XCIII, pp. 126–150, 1923.

[13] Herdan, G.: Causes of Excess Male Mortality in Man. *Acta Genetica,* 1952, III, pp. 351–375.

[14] Madigan, F. C.: Are Sex Mortality Differentials Biologically Caused? Milbank Memorial Fund *Quarterly,* April, 1957, XXXV, pp. 202–223.

[15] From a computation similar to that described in Footnote 10, it can be shown that the age group 15–24 made only a minor contribution. The greatest contributions to increases in the sex mortality ratio at all ages (not age-adjusted) were by the age groups 45–54, 55–64, and 65–74.

from such degenerative diseases on biological grounds, it is difficult to explain trends in sex differences on this basis.

In general, an examination of causes of death responsible for increases in sex differentials in mortality suggests that two kinds of forces are at work. On the one hand, social, medical, and public health advances have caused death rates for certain female diseases, or diseases with low sex mortality ratios—tuberculosis, diseases associated with childbearing, cancer of the uterus, and diseases associated with high blood pressure—to decline. At the same time, some factors have caused increases in male death rates for motor vehicle accidents, lung cancer, and coronary heart disease. It would seem important to identify those factors responsible for increases in death rates, and to counteract the growing imbalance in mortality patterns.

Summary

1. In the United States, during the period 1920 to 1958, the ratio of white male to white female age-adjusted death rates increased from 1.08 to 1.60.

2. During the period 1929–31 to 1956–58, the largest increases in sex mortality ratios occurred in the age groups 15–24 and 45–64.

3. Trends in death rates for tuberculosis, maternal mortality, and motor vehicle accidents were primarily responsible for increases in the age group 15–24.

4. Trends in death rates for cancer of the lung, cancer of the uterus, coronary heart disease, and diseases associated with high blood pressure were important causes for increases in the age group 45–64.

5. Two kinds of trends in death rates have affected sex mortality ratios: declines in causes of death importantly affecting females (tuberculosis, maternal mortality, cancer of the uterus, and diseases associated with high blood pressure), and increases in male death rates for motor vehicle accidents, lung cancer, and coronary heart disease.

6. While increases in sex mortality ratios are frequently attributed to biological factors, an examination of the causes of death responsible, in the age groups where the greatest increases have occurred, suggests that environmental factors may be considerably more important.

Appendix Table A. Average annual death rates for selected causes per 100,000 population, white persons, age groups 15–24 and 45–64, for the periods 1929–1931, 1939–1941, 1949–1951, 1956–1958.

Cause of Death[2]	Age Group 15–24								Age Group 45–64[1]							
	1929–1931		1939–1941		1949–1951		1956–1958		1929–1931		1939–1941		1949–1951		1956–1958	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
All Causes	300.7	258.3	201.1	139.4	154.9	71.9	151.1	57.1	1776.6	1369.5	1677.0	1124.0	1524.6	851.5	1447.1	720.0
Tuberculosis, All Forms (001–019)	40.7	67.0	17.2	27.8	4.7	6.9	.5	.5	111.1	57.6	90.7	32.9	55.6	13.7	20.6	4.8
Syphilis and its Sequelae (020–029)	1.4	1.5	.8	1.1	.2	.1	.1	0	44.9	13.1	38.6	10.2	13.3	3.4	5.2	1.4
Typhoid Fever (040)	6.8	4.6	1.3	1.0	0.0	0.0	0	0	3.5	2.2	1.0	.5	.1	0.0	0.0	0
Dysentery, All Forms (045–048)	.2	.2	.2	.1	.1	0.0	0	0	1.1	1.0	.6	.6	.3	.2	.1	.1
Diphtheria (055)	.7	.8	.1	.2	0.0	.1	0	0	.4	.7	.2	.2	.2	0.0	0.0	0
Whooping Cough (056)	0.0	.1	0.0	0.0	0.0	0.0	0	0	0.0	0.0	0.0	0.0	0.0	0.0	0	0
Meningococcal Infections (057)	5.4	1.9	.6	.3	.4	.2	.2	.1	1.6	.9	.4	.3	.3	.2	.1	.1
Acute Poliomyelitis (080)	1.5	.8	1.1	.5	2.5	1.7	.3	.4	.2	.1	.1	.1	.2	.1	.1	0.0
Measles (085)	.6	.6	.3	.3	.1	.1	.1	.1	.2	.4	.1	.2	0.0	0.0	0	0
Malignant Neoplasms (140–205)	4.9	3.9	6.0	4.4	10.7	7.4	10.6	6.9	199.6	283.9	222.0	274.4	255.3	258.3	273.7	239.8
Diabetes Mellitus (260)	2.9	2.8	2.0	2.3	.9	1.3	.9	1.3	32.6	58.4	33.7	62.3	18.7	28.9	17.5	21.5
Vascular Lesions Affecting Central Nervous System (330–334)	1.8	1.8	1.7	1.4	1.5	1.3	1.9	1.5	142.6	135.6	114.3	110.8	105.9	96.2	88.2	68.8
Rheumatic Fever (400–402)	1.9	2.1	1.3	1.2	1.2	1.2	.4	.3	2.3	2.1	.5	.6	.9	.6	.7	.5
Diseases of Heart (410–443)	18.7	20.3	13.7	12.4	6.4	5.2	4.3	3.3	419.0	279.4	566.4	277.1	687.7	268.9	670.1	224.0
Hypertension without Mention of Heart (444–447)	0.0[3]	0.0[3]	0.0	.1	.2	.2	.1	.1	.6[3]	.6[3]	1.5	1.4	9.4	7.0	6.5	4.5
General Arteriosclerosis (450)	0.0[3]	0.0[3]	0.0	0.0	0.0	0.0	0	0	15.1[3]	9.9[3]	8.8	5.7	6.7	3.6	5.9	3.0
Chronic and Unspecified Nephritis and Other Renal Sclerosis (592–594)	5.4	6.1	4.0	4.0	2.9	2.1	2.2	1.5	143.6	127.9	103.1	82.8	20.3	15.3	10.8	7.5

Influenza and Pneumonia, Except Pneumonia of Newborn (480–493)	29.5	21.6	10.4	8.2	2.9	2.3	3.3	3.0	147.6	100.5	77.7	45.2	30.0	13.1	30.4	12.9
Ulcer of Stomach and Duodenum (540, 541)	1.5	.6	.9	.2	.3	.1	.2	.1	23.8	6.3	28.1	4.4	19.9	3.3	18.1	3.8
Gastritis, Duodenitis, Enteritis, and Colitis, Except Diarrhea of Newborn (543, 571, 572)	.7	1.0	.7	.7	.6	.6	.5	.4	4.0	3.8	2.2	2.0	3.0	2.2	3.7	2.5
Cirrhosis of Liver (581)	.3	.3	.3	.4	.2	.4	.2	.3	25.5	12.2	29.7	12.6	32.3	14.5	39.1	16.7
Acute Nephritis and Nephritis with Edema, including Nephrosis (590, 591)	1.4	1.5	1.0	.8	1.0	.6	.5	.3	5.3	4.0	2.8	1.5	2.8	1.9	1.7	1.1
Deliveries and Complications of Pregnancy, Childbirth, and the Puerperium (640–689)	—	37.6	—	18.2	—	5.2	—	2.4	—	1.3	—	.6	—	.1	—	.1
Congenital Malformations (750–759)	.7	.6	2.1	1.1	2.1	1.6	2.6	1.9	.1	.1	.6	.5	2.7	2.1	3.0	2.4
Symptoms, Senility, and Ill-Defined Conditions (780–795)	1.4	1.7	1.1	1.2	1.1	.7	1.4	.7	15.4	8.0	15.8	6.2	13.1	4.6	12.8	4.5
Motor Vehicle Accidents (E810–E835)	43.3	12.5	48.5	12.9	57.0	12.4	66.9	15.2	53.2	18.9	51.7	15.3	35.7	12.4	33.8	12.8
Accidents, Except Motor Vehicle (E800–E802, E840–E962)	56.5	8.7	39.2	6.0	34.9	5.3	32.6	4.4	101.1	23.9	73.7	19.1	54.8	13.3	45.6	11.8
Suicide (E963, E970–E979)	9.2	6.1	8.4	4.0	6.7	2.5	6.7	2.1	60.8	13.9	47.2	13.5	37.3	10.3	35.2	9.8
Homicide (E964, E980–E985)	8.7	3.4	4.0	1.3	3.8	1.3	3.9	1.5	11.0	1.8	6.4	1.4	5.1	1.5	4.4	1.6
All Other Causes	54.6	48.2	34.2	27.3	12.5	11.1	10.7	8.8	210.4	201.0	159.1	141.6	113.0	75.8	119.8	64.0

0.0 rate is more than 0 but less than .05; — means not applicable.

[1] Death rates are age-adjusted in 10-year intervals to the 1940 United States population.

[2] Numbers in parentheses are 1948 International List numbers. For corresponding rubrics for years prior to 1948, see *Vital Statistics—Special Reports*, Vol. 43, National Office of Vital Statistics.

[3] Average of 1930–31.

SOURCE: *1929–1951; Vital Statistics—Special Reports*, Vol. 43, National Office of Vital Statistics. Deliveries and complications of pregnancy, childbirth, and the puerperium: from annual volumes of National Office of Vital Statistics.
1956, 1957; VITAL STATISTICS OF THE UNITED STATES, 1956, Volume I, and 1957, Volume I, National Office of Vital Statistics.
1958; Unpublished data from National Office of Vital Statistics.

Appendix Table B. Average annual death rates for malignant neoplasms and diseases of the heart per 100,000 population, white persons, age group 45–64,[1] for the periods 1949–1951 and 1956–1958.

Cause of Death[2]	1949–1951		1956–1958	
	Male	Female	Male	Female
Malignant Neoplasms (140–205)	255.3	258.3	273.7	239.8
Mouth, Pharynx, and Esophagus (140–148, 150)	17.4	3.8	18.1	4.0
Stomach (151)	32.1	15.7	22.1	10.7
Intestines and Rectum (152–154)	36.8	37.8	34.9	33.7
Pancreas (157)	14.1	8.2	16.5	8.8
Lung (162, 163)	55.1	8.8	78.8	10.0
Prostate (177)	10.3	—	9.6	—
Kidney (180)	7.0	3.4	7.6	3.3
Bladder (181)	9.2	3.2	8.0	2.6
Breast (170)	.6	56.6	.5	57.6
Uterus (171–174)	—	43.6	—	33.0
Lymphoma (200–203)	13.1	8.2	16.3	10.0
Leukemia (204)	10.1	7.2	10.7	7.4
Other Cancer	49.5	61.7	50.6	58.6
Diseases of the Heart (410–443)	687.7	268.9	670.1	224.0
Rheumatic (410–416)	27.6	25.9	25.5	24.8
Coronary (420)	519.9	146.9	561.6	146.9
Myocardial Degeneration (421–422)	50.5	28.2	24.7	13.5
Other Diseases of the Heart (430–434)	26.9	11.2	20.5	8.2
Hypertensive (440–443)	62.9	56.6	37.7	30.7

— means not applicable.

[1] Death rates are age-adjusted in 10-year intervals to the 1940 United States population.

[2] Numbers in parentheses are 1948 International List numbers.

Source: *1949, 1951, 1956, 1957: Vital Statistics of the United States*, Volume II for each year specified, National Office of Vital Statistics.
1950: Vital Statistics of the United States, 1950, Volume III, National Office of Vital Statistics.
1958: Unpublished data from National Office of Vital Statistics.

IMPLICATIONS OF PROSPECTIVE UNITED STATES POPULATION GROWTH IN THE 1960S

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I. DEMOGRAPHIC BACKGROUND AND PROSPECTS SUMMARIZED

THE United States is entering the third decade of a demographic revolution of profound significance, which has already contributed much to transform our national position, outlook, and problems.[2]

The 1940s witnessed an unprecedented rise in the prevalence of the married state, a decline in the median age at first marriage, a marked rise in the "general fertility rate" (number of live births per 1,000 women aged 15–44),[3] and first one and then another so-called "baby boom."[4] These were proximately responsible for our wholly unexpected population upsurge.

These developments surprisingly continued in the 1950s, though at a slower pace. Births, instead of declining, flooded to a new high average of 4¼ million a year in 1956–1959. Hence our vigorous population increase was remarkably sustained through the past decade. It is hard to exaggerate the transformation of our population position and outlook between 1940 and 1960.

In the 1960s our population growth is likely to continue

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[2] The data on which my statements are based are mainly those of the Departments of Commerce, Labor, and Health, Education, and Welfare. Many of these are summarized in the STATISTICAL ABSTRACT OF THE UNITED STATES, 1959, HISTORICAL STATISTICS OF THE UNITED STATES (1960 edition), HEW TRENDS, 1960 edition, and *Economic Reports of the President*. Most of the details are in *Current Population Reports* (Series P-25, No. 187, Nov. 10, 1958, is especially important); in *Vital Statistics of the United States* and other publications of the National Office of Vital Statistics, Public Health Service; and in publications and press releases of the Office of Education, also in the Department of Health, Education, and Welfare.

[3] Time series of *crude* birth rates, marriage rates, and death rates (i.e., number per 1,000 population) are misleading because of radical changes since 1940 in the age composition and marital status of the population.

[4] The term "boom" is misleading for an upswing which is not followed by a "bust" or substantial recession. The terms "bomb" and "explosion" are still more inapt for even rapid population growth movements.

vigorous—not rapid, as it was in 1790–1860[5]—if only we escape catastrophic destruction of human and natural resources and severe damage to plant, animal, and human fertility. The numerical gain will probably at least exceed the record-large 28 million increase in the 1950s,[6] by a margin that may be small or considerable. The prospective rate of gain is also unpredictable. It may be slightly below that of the past decade—about 18.5 per cent—the highest since 1900–1910; but it will vary from year to year and will probably be slower in the first half of the 1960s than in the second.

A quick cartographic summary of selected data and projections is given in Figures 1–5.[7] Figure 1 is an updated version of Chart 15 in the present writer's pamphlet, THE POPULATION UPSURGE IN THE UNITED STATES (Food Research Institute, December 1949). Plotted on a semi-logarithmic or ratio scale, it shows our population growth in long perspective. One can observe the virtual stability of the growth rate in 1800–1860, its persistent tapering off in 1860–1930, the severe slump in the 1930s, and the subsequent sustained upsurge.

A few representative projections or "forecasts" published in 1920–1946 serve to bring out the unexpectedness of the reversal in 1940–1960. The highest curves, extending only to 1980, show

[5] If the 3.02 per cent average annual rate of increase in that period had continued through the next century, our population now would be over 600 million. California's population increased in 1860–1960 at a fairly sustained rate still more rapid, and higher than in almost any country today.

[6] Three "illustrative projections" for 1960, the first official ones to take account of the population upsurge, were released by the Bureau of the Census just ten years ago. *Current Population Reports,* Series P-25, No. 43, Aug. 10, 1950. The highest of these projections will prove substantially correct, but it slightly underestimated the increase in the decade. Unlike later ones, these made allowance for census undercounts of children under 5.

On census undercounts and the results of errors in age reporting, see Coale, Ansley J.: The Population of the United States in 1950 Classified by Age, Sex, Color—A Revision of Census Figures. *Journal of the American Statistical Association,* March 1955, L, 16–54; and Smith, T. Lynn: A Demographic Study of the American Negro. *Social Forces,* March 1945, XXIII, 379–87. The undercount is relatively largest in children under 5 and in nonwhites in various age groups. Conceivably the coverage of the 1960 census will be more nearly complete and less inaccurate on age data than were those of 1950 and 1940. If so, the calculated population increase will overstate the true increase, and comparisons of age groups will be somewhat distorted.

[7] Thanks are due Patricia Cedarleaf of the Food Research Institute for drafting the charts.

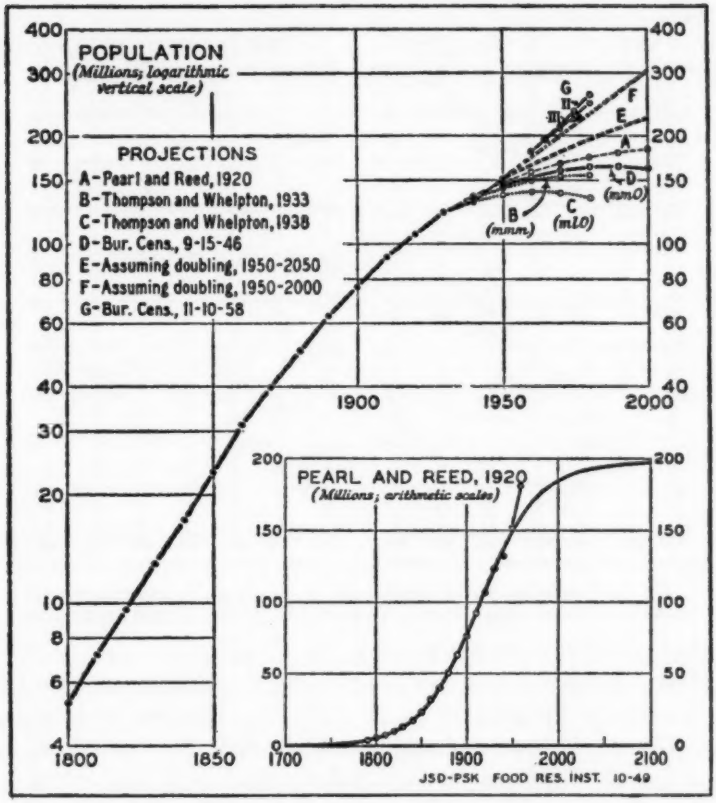


Fig. 1. United States population by decades, 1800–1960, with selected projections.

two of the four latest official projections, published by the Bureau of the Census November 10, 1958. Currently, Series II and III look the more credible, but neither can be wholly trusted.

The inset chart shows the Pearl and Reed 1920 logistic curve, plotted on an arithmetic scale. The fit with decennial census data was fairly close in 1920–1950, but the 1960 census figure will be far above the curve. Its future course is wholly unbelievable, since its most basic assumption has become unten-

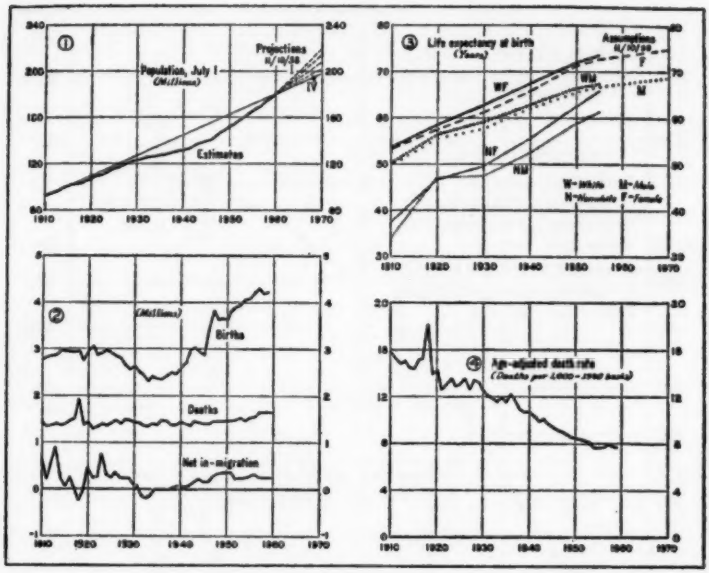


Fig. 2. Population, births, deaths, net in-migration, life expectancy, and age-adjusted death rates, United States 1910–1960, and selected projections.

able (see POPULATION UPSURGE, pp. 72–73).

Figures 2 and 3 together contain eight subcharts of population data and vital statistics for 1910–60, with some projections to 1970 of which no endorsement is implied. Special attention is called to the separate curves for whites and nonwhites in Figure 2, subchart 3 and Figure 3, subcharts 2 and 4. Attention is also called to increases in marriage and fertility rates after 1940 reflected in Figure 3, subcharts 1 and 2.

Figures 4 and 5 together contain nine subcharts, for different time periods, illustrating a number of points made in the first two sections of the paper. Special attention is called to Figure 4, subcharts 1, with its startling projections of births, and 2, showing the notable "echo effects"; Figure 5, subcharts 1 and 2, showing the prospective growth of highly significant age groups under 35; and 4, showing the relative size of age groups 18–64 and the sum of younger and older groups.

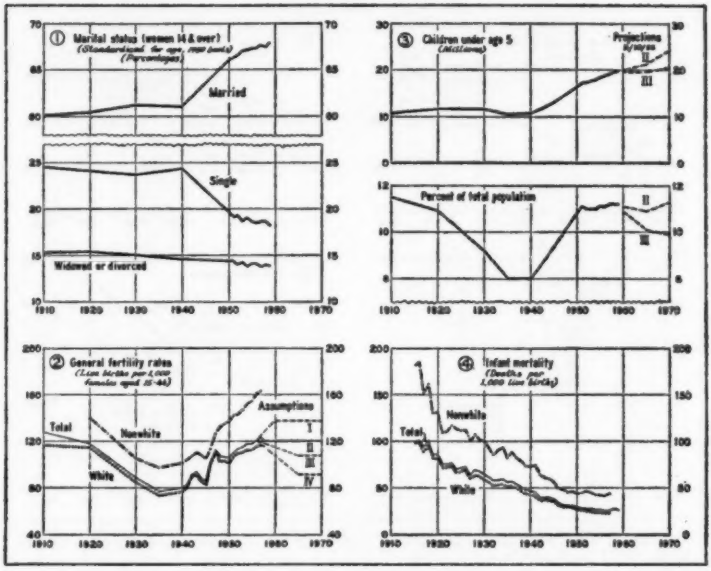


Fig. 3. Marital status, fertility rates, children under 5, and infant mortality rates, United States 1910–1960, and selected projections.

We cannot safely forecast the course of the fertility rate or the number of births.[8] Yet we can reasonably expect that births in the 1960s will at least exceed the 40.5 million in 1950–1959, for three reasons: (1) there is no sign that our strong preferences for the married state, and for early marriage, will weaken soon;[9] (2) the number of women reaching age 20, and the

[8] The course of births in the 1950s bore no resemblance to any of the three official projections published in August 1950, and the total for fiscal years 1950–1959 was 2.1 million (5 per cent) above the sum of the births indicated by the high projection for the same 10-year period. For 1951–1960 the excess will be slightly larger.

[9] Percentages of women in selected age groups, married and single.

Year	Married				Single (Never Married)			
	18–19	20–24	25–34[a]	18–34[a]	18–19	20–24	25–34[a]	18–34[a]
1940	21.7	51.3	77.1	61.9	77.8	47.2	18.9	35.3
1950	31.6	66.2	85.5	74.0	67.9	31.6	10.8	22.6
1959	33.7	69.7	88.6	76.7	65.9	28.5	8.2	20.7

[a] Not standardized for age.

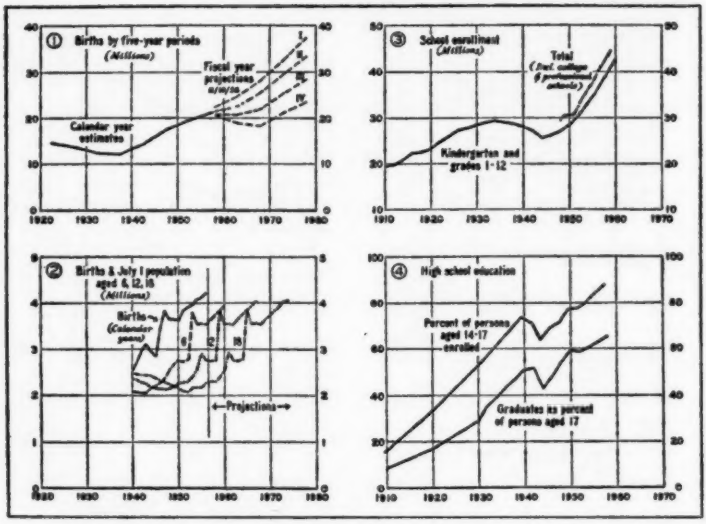


Fig. 4. Births, school enrollment, and high school education, United States 1910–1960, and selected projections.

numbers in the most fertile age groups, will grow impressively, especially in the second half of the decade; and (3) the prospects are good for avoiding a severe and protracted depression that might seriously curtail marriages and fertility for a time. Conceivably, births may prove even more numerous than 52.7 million in fiscal years 1961–1970—the figure implied in the highest of the four 1958 official projections; but, considering the recent height of the fertility rate, the Series I assumption that it will average 10 per cent above the 1955–1957 level (120.5) now seems too liberal.

These are the areas of greatest demographic uncertainty as we look a decade ahead—the limit of my assigment. Those in which projections have a solid basis are relatively much more important.

Though much in the unfolding future is obscure, we can have confidence in the statistics of past births—by far the most significant demographic series. We can expect the birth curve to continue to be echoed in curves for one-year age groups, since

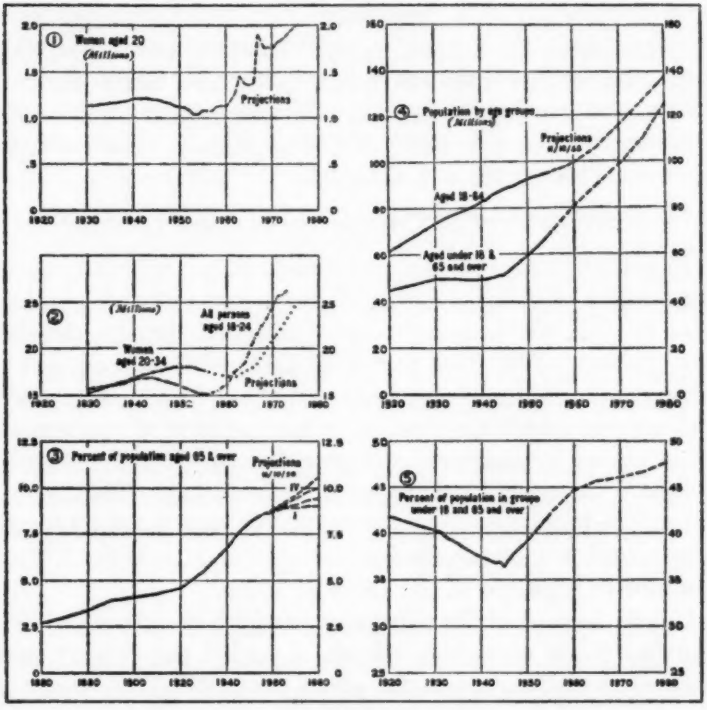


Fig. 5. Trends in the growth of significant age groups in the United States 1860–1960, and projections.

infant, child, and youth mortality rates have fallen very low and net in-migration is a small element in our population growth. We can put substantial trust in 10-year projections of most age groups over age 10, and expect their total in mid-1970 to be within 2–5 per cent of 167.5 million. The numbers aged 14 and over will increase by some 24 million in the 1960s, more than in the two decades 1940–60.[10] Such facts are of high significance for business, economic, and social policy and planning.

The most important population development in this momentous decade will be the growing older of persons now living who

[10] See: Labor Resources in the Sixties. Federal Reserve Bank of Chicago, *Business Conditions*, July 1960, pp. 10–16.

were born after 1939. Because of this, and continuing noteworthy gains in health and educational attainment, the effective increase in population will be larger in terms of needs, wants, and productive capacity than in mere numbers. But striking changes will surely occur in highly significant age groups in this decade and the next.

II. Special Aspects of Coming Population Developments

In the 1950s there was a remarkable 50 per cent increase in the number of children in kindergarten and elementary school ages (5–13), which unexpectedly continued through the decade. Between 1949–1950 and 1959–1960, while the number of persons in high-school ages increased about 33 per cent, public and private school enrollments in kindergarten through grade 12 increased by nearly 49 per cent, from 28.7 million to 42.7 million.[11] In view of the course of births in 1945–1960, further sizeable increases are in clear prospect. No peak in school enrollments, such as a decade ago was expected to come in the 1950s,[12] can now be expected in the 1960s or 1970s.

Chiefly because of the rising appetite for high-school training, enrollments in secondary schools exceeded the prewar peak early in the 1950s, and in the school year 1957–1958 nearly 88 per cent of all Americans aged 14–17 were enrolled in school.[11] Now, in consequence of the great increase in births in 1946–1947, an upsurge in the number of 13-year-olds is causing a marked rise in high-school enrollments which will continue in the 1960s and 1970s.

The teen-age population (ages 13–19) had declined in the 1940s in response to the fall in births in 1925–1935, and increased only moderately in the 1950s, in response to birth increases in 1935–1945 and to reduction in infant, child, and youth

[11] Office of Education: Progress of Public Education in the United States of America, 1959–60 (Washington, July 1960), pp. 10–11.

[12] Office of Education projections for 1950–60, published in *School Life*, May 1950, xxxii, 116 (quoted in Joint Committee on the Economic Report, The Sustaining Forces Ahead, 1952, p. 77) had indicated peaks in total and elementary enrollments (excluding kindergarten pupils) late in the 1950s, at 32.1 and 25.5 million respectively, with 1960 figures slightly lower.

mortality since 1935. A much sharper rise, irregular in character, is now in progress. Now numbering about 20 million,[13] teen-agers will increase by about one-third in this decade, to about 70 per cent above the low of mid-1950.

The number reaching age 18 declined to a postwar low of 2.1 million in fiscal 1952. It will jump sharply in 1964–1965, reflecting the first post-war peak of births in 1946–1947, and will average nearly as large in the rest of the 1960s. The college age group proper (18–21) will grow relatively fast in this decade, from about 9.6 million to 16.3 million, and will nearly double in 1955–1975.

The age group 18–24 is especially significant, since it includes the great majority of those enrolled in colleges and universities, provides most of the newly married couples, and furnishes most of the first-born children. In the early 1960s this age group will increase by an average of about one-half million a year, and in the second half of the decade by about a million a year. In 1970 it will number about 25 million, 10 million more than in 1957.

The number of women aged 18–24 or 20–24, age groups of special significance for first marriages and first births, declined in the 1950s but will increase by over 50 per cent in the 1960s, the more rapidly in the second half of the decade. The number of women in the most fertile age groups, 20–34, declined slightly in 1950–1955 and further in 1955–1960, but will increase nearly 5 per cent in 1960–1965 and about 17½ per cent in 1965–1970.

The population aged 25–44 will increase by only about 3 per cent in the 1960s, as compared with a total population increase probably ranging from 16.3 per cent (Series III) to 19.0 per cent (Series II). The latest projections for this age group in 1960 and 1970 are 46.8 and 48.2 million respectively.

The percentage of the population in the most productive age groups, 20–64, rose from 51.7 in 1900 to a peak of 59.5 in 1945, then declined to about 52.5 in 1960, in consequence of the post-

[13] My estimate, since the Census Bureau does not publish figures for this odd age group.

1940 flood of births and the swelling of the numbers of older people.[14] The decline will continue at a slower pace, probably to about 50.5 in 1970. It is remarkable that American levels of consumption, education, and living have notably risen[15] while a significant "dependency ratio" (the sum of percentages under 20 and over 64) has risen in 1945–1960 from 40.5 about 47.5.

Projections of the labor force have mostly proved too conservative.[16] The chief economist of the National Planning Association late in 1952, however, quite closely forecast the actual figure for 1960 at 72.5 million. The NPA staff "judgment projection" in October 1959 for 1970 (85.9 million)[17] must therefore command respect, though slightly higher figures are given by the Bureau of Labor Statistics (87.1 million)[18] and the two higher projections of the Census Bureau (87.3) published earlier in 1959.

The big uncertainties still concern labor participation rates, especially for women.[19] The NPA and BLS figures imply a total increase of 13.4–13.5 million during the 1960s comparing with one of 7.8 million in the 1950s. Some such striking increase will

[14] Based on official data in HEW TRENDS, 1960 Ed., p. 1. These ignore persons missed by census enumerators; the series of percentages would be slightly lower if we could adjust for these omissions.

[15] ECONOMIC REPORT OF THE PRESIDENT January, 1960, Appendix C: Statistical Tables Relating to the Diffusion of Well-Being. Per capita consumption expenditures in 1959 dollars, a crude but useful general measure, rose from $1,470 in 1946 to $1,760 in 1959, an increase of nearly 20 per cent in 13 years. Mean personal income (after tax) per family, in 1959 dollars, rose 22½ per cent in 1947–1959. *Ibid.*, p. 132.

[16] Late in 1949, Slichter forecast the labor force in 1980 at 72.5 million. Slichter, S. H.: How Big in 1980? *Atlantic Monthly*, November 1949, pp. 39–43, esp. p. 39. This figure is being reached in 1960. The latest Bureau of the Census projections for 1980, published in December 1958, range from 101.5 to 104.8 million.—STATISTICAL ABSTRACT OF THE UNITED STATES, 1959, p. 207. Late in 1952 the Bureau of the Census had projected the labor force at about 89 million in 1975. *Current Population Reports*, Series P-50, No. 42, Dec. 10, 1952. Its latest projections for 1975 range from 93.3 to 95.7 million.

[17] Colm, Gerhard: *The National Economy in* 1960. (NPA Planning Pamphlet 81, Washington, Dec. 1952), pp. 15–17, and NPA Staff Report, *Long-Range Projections for Economic Growth* (NPA Planning Pamphlet 107, Oct. 1959), p. 6.

[18] HEW TRENDS. 1960 Ed., p. 12, and Bureau of Labor Statistics, *Population and Labor Force Projections for the United States, 1960 to 1975* (Bulletin 1242, June 1959).

[19] See Bancroft, Gertrude: Factors in Labor Force Growth. ASA, *Proceedings of the Social Statistics Section, 1959* (Washington, 1960), pp. 29–33.

doubtless occur, but various uncertainties (e.g., regarding the extent of shortening the workweek, changes in the relative importance of part-time work,[20] and employment of older men) cast doubt on precise forecasts. The most marked increases will almost surely be in workers under age 25 (perhaps 46 per cent) and in workers aged 45–64 (perhaps 20 per cent), while "the number of women workers will increase at nearly twice the rate for men."[21] The median age of the labor force, which has risen strikingly in the past two decades, will begin an impressive fall in the 1960s.

Completion of childbearing at earlier ages (many of them under age 30)[22] tends to release more mothers from pressing family duties in middle age, permitting them to enter the labor force.[19] It is striking that the number of married women over 35 in the labor force rose from 2.1 million in 1940 to 4.9 million in 1950 and 8.1 million in 1959, implying about a four-fold increase in two decades. The unexpectedly large net increase in the total labor force in 1940–1960 was due mainly to this; and its prospective continuation is largely responsible for the official projection that by 1970 there will be about 30 million women workers, 25 per cent more than in 1960.

The persistent tendency to prolong one's schooling, reinforced by evidence that more education tends to increase individual earnings,[23] is raising the median age of entrance into the full-time labor force. Of this we have no precise measure. The numbers reaching age 18 will be much higher in 1961–1964, and sharply higher in 1964–1970, than in most of the 1950s.

[20] *Federal Reserve Bulletin*, May 1960, p. 469.

[21] United States Dept. of Labor: MANPOWER: CHALLENGE OF THE 1960s (Washington, 1960).

[22] Glick gives the median age of wives (in years) at the birth of the last child as follows: 1890—31.9; 1940—27.1; 1950—26.1. Glick, Paul C.: The Life Cycle of the Family. *Marriage and Family Living*, Feb. 1955, XVII, 4.

[23] The 1950 Census of Population provided the basis for computing, on a 3½ per cent sample, the median income by years of school completed, for various age groups in 1949. See STATISTICAL ABSTRACT OF THE UNITED STATES, 1959, p. 110. Similar data from the 1960 Census are likely to be still more impressive. More extensive but different data for 1957 are broadly confirmatory. See *Current Population Reports*, Series P-60, No. 27, Apr. 1958, esp. pp. 10–11.

This will be followed, with some lag, by a heavy flow of young entrants into the full-time labor force, and its effect will be increasingly felt through the decade 1965–1975. The United States Department of Labor estimates that 46 per cent of the increase in the total labor force in 1960–1970 will be workers under age 25, and that the increase in this group will be about 10-fold that in 1950–1960.[21] The biggest increases in opportunities for employment will be in professional and technical jobs, and there will be little change in the number of jobs for the unskilled, who have been most subject to extensive unemployment in the 1950s.

The younger persons who will enter the labor force in increasing numbers in the late 1960s and 1970s will have had much more schooling than those who will be leaving the labor force in these years,[24] and illiteracy will be low even among nonwhite entrants. Most of those retiring will have completed well under 8 years of schooling, while something like two-thirds of those entering will have completed high school, and a sizeable fraction of these will have completed four years of college. Granting that exposure to schooling does not ensure educational achievement, and that there are quality deficiencies in schooling, there is no doubt that the educational level of the labor force is rising significantly.

The number of persons in age groups 65 and over (commonly miscalled "the aged") has been and is continuing to increase faster than the total population,[25] but the percentage in these age groups has risen only from 8.1 in 1950 to about 8.8 in 1960 and is likely to be 9.1–9.4 in 1970 and under 10 in 1980. The 1958 official projections indicate that in 1950–1980 the total

[24] See: Projections of Educational Attainment in the United States, 1960–1980. *Current Population Reports,* Series P-20, No. 91, Jan. 12, 1959 (summarized in STATISTICAL ABSTRACT OF THE UNITED STATES, *1959,* p. 112), and Medsker, Leland S.: THE JUNIOR COLLEGE: PROGRESS AND PROSPECTS. McGraw-Hill, New York, Mar. 1960.

[25] See the valuable volume in the Census Monograph Series by Sheldon, Henry D. and Tibbitts, Clark: THE OLDER POPULATION OF THE UNITED STATES. Wiley, New York, 1958; an illuminating article by Civic, Miriam I.: Recent Trends in Income of Older Persons. In *Conference Board Business Record,* Aug. 1960, XVII (8), 10–13; and data in STATISTICAL ABSTRACT OF THE UNITED STATES, *1954,* p. 263, *ibid., 1956,* p. 259, and *ibid., 1959,* p. 270.

number will double, but that those aged 65–69 will increase by 75 per cent while those aged 85 and over will increase by over 200 per cent.

So great has been the increase in the number of young people since 1940 that the median age of the population, which had risen persistently for 150 years, reached a peak in the early 1950s (30.2 years) and will decline for at least another decade or two. In this significant sense, our Nation is now growing *younger*, not older.[26] Moreover, increasing recognition is rightly given to the progressive "youthening of the elderly," which leaves growing fractions of those in age groups 65–74 competent physically and mentally. Though the contributions made by older people are not readily measured—many of them important though nonmaterial—they are surely far below potentials. Here is a significant "new frontier" on which pioneering is under way.

The number of deaths per year, which had risen very slightly in 1910–1950, trended moderately upward in the 1950s, rising from 1.45 million in 1950 to about 1.65 million in 1959. Some such uptrend can be expected to continue through the 1960s and beyond. Yet it is confidently expected that the age-adjusted death rate will resume its long-term downtrend (the recent interruption, in 1954–1959, had a precedent in the 1920s). Life expectancy at birth is expected to continue to rise, at a slowing pace because the levels for various age groups are now so high. Estimates underlying the 1958 official population projections suggest a rise for females from 72.9 years in 1955 to 76.0 in 1975–1980 and to 77.1 in the year 2000.[27] These may prove conservative, especially if widely anticipated "breakthroughs" are made in coping with cancers and heart diseases,

[26] Notestein, Frank W.: As the Nation Grows Younger. *Atlantic Monthly*, October 1957, pp. 131–36. "This trend to a younger average age is new in the Western world." It is of slight significance that the mean age of the population is expected to change little over the next 20 years.

[27] 1958 *Projections*, p. 12. The same source gives corresponding figures for males as 66.7, 69.8, and 71.3, and (p. 13) the following projections of the crude death rate by quinquennia for projection Series II: 1950–55—9.5; 1955–60—9.2; 1960–65—8.9; 1965–70—8.7; 1970–75—8.4; 1975–80—8.1.

which have greatly increased in prevalence as life expectancy has risen.[28]

It is important also to note the rising proportion of nonwhites. In 1900 they made up about 12.1 per cent of the population. By 1920 the percentage had fallen to 10.3, and it remained at about this level through 1949. In the 1950s it rose to 11.0 in 1957 and 1958. This rise, largely the result of the much higher fertility of nonwhite women and the more marked improvement in life expectancy of nonwhites,[29] will almost certainly continue in the 1960s.

Finally, a substantial redistribution of our population has accompanied the vigorous growth of the postwar period. Three types deserve emphasis. (1) Important state and regional shifts have taken place. The largest absolute increases in state populations in 1950–1960 were in California (over 5 million), Florida, New York, Texas, and Ohio (1.8 million), in this order, and the largest relative increases in Florida, Nevada, Alaska, Arizona, and California, in this order, while West Virginia, Arkansas, and Mississippi appear to have lost population. (2) Most of the recent growth has been concentrated in the "standard metropolitan statistical areas," reflecting the notable suburbanization of our people, while central cities and truly rural areas have tended to lose population relatively, and in some instances absolutely. (3) Employment in agriculture, like the farm population, has continued to decline, while government, trade, and service occupations have continued to make large gains in employment. Though the 1960s will not faithfully follow the pattern of the 1950s in these respects, further redistribution of population of all three types bids fair to be substantial in this decade.

The prospective population developments in the 1960s have many significant implications and evoke many pertinent observations. I have time to present briefly only a few of each.

[28] See: Recent and Future Mortality Trends. In Metropolitan Life Insurance Company *Statistical Bulletin*. June 1960, pp. 1–3.

[29] HEW Trends. 1960 Ed., pp. 2–4, and *Vital Statistics of the United States*, e.g., 1955 (Washington, 1957), I, Table AC.

III. Leading Implications

1. The aggregate demand for consumption goods and services in the 1960s—on the reasonable assumption that earnings and total purchasing power will insure conversion of wants into effective demand—will rise significantly more than in the 1950s, because those born since World War II will be a decade older, their educational level will be higher, and disposable family income will be generally larger.

2. Especially pronounced will be the demands for more educational facilities and qualified personnel. Our ability to meet these demands will be under continuing strain, even though we count as our most vital investment, that in "human resources," what we spend on the schooling of our children, youth, and young adults.[30] (a) The sustained flood of births in the 1950s insures continuing expansion, if at a slowing pace, in needs for elementary education. (b) The expansion will be much sharper in requirements for secondary education, primarily because the numbers aged 14–17 are increasing strikingly, but also because economic incentives keep sharpening appetites for more schooling.[31] (c) For similar reasons, perhaps after a moderate time lag, the demands for facilities and personnel for junior college, 4-year college, and university education will rise even more sharply.

3. The marked increase in the number of teen-agers in the 1960s (probably by about one-third) will not only expand their aggregate demands for all sorts of nondurable goods, cars, schooling, recreational facilities, and part-time jobs, but will also intensify baffling problems of traffic congestion, automobile accidents, and juvenile crime.

4. For lack of education and experience, adverse discrimination, and other reasons, the capacities of nonwhites are not being adequately developed and used to the advantage of the Nation, and rates of unemployment, illegitimacy, and crime

[30] Atwater, Thomas V., Jr.: Education: Key Economic Problem. *In* Problems of Economic Development (CED, New York, May, 1958), II, pp. 325–32.

[31] Some data are given in Statistical Abstract of the United States, 1959, p. 110.

are much higher among them. The relatively rapid increase in the nonwhite population, it is necessary to add, must tend to retard their economic and social progress. Disparities between whites and nonwhites are still wide, though they have been very significantly narrowed. Faster progress in this direction will be more urgent in the 1960s, when there will be a marked enlargement of the group of Negro youth.

5. Among the major tasks of the near future will be the smooth absorption of a much enlarged flow of young entrants into the full-time labor force, and appraising and coping with its repercussions on other components of the labor force, on hours of labor, and on part-time employment.

6. Our continuing population upsurge, coupled with the rise in per capita investment required to support our high and rising level of living, virtually assures increasing demands for investment capital in the United States, while the pressure for American investments to supplement limited supplies of domestic capital in the developing nations is increasing because of their population increase and rising "aspirations." Odell recently concluded:

> The demand for investment capital in and from the United States through the next twenty years will be so strong that the greatest economic problem will be to limit the amount of capital investment to a level which can be met primarily from real savings.[32]

Some such emphasis is justified, even if one cannot wholly endorse this assertion.

7. In the second half of the 1960s we can confidently expect a major upswing in family and household formations, though our ability to forecast their timing and extent is still weak. In consequence, a "housing boom" of large proportions will presumably start before the end of the decade and continue in the

[32] Odell, William R. (vice president and director, International Harvester Company): A World Wide Shortage of Investment Capital. *In* PROBLEMS OF ECONOMIC DEVELOPMENT, *op. cit.*, II, 89–93.

1970s.[33] The prospective expansion will exert pressures to enlarge the building industry's labor force, materials, supply, and financing, when other construction requirements will also be making heavy demands. These pressures will also aggravate the already difficult problem of providing and financing investment in local public facilities.[34]

8. Expansion of suburban residential and light industrial areas, together with the decentralization of heavier industry and modern industrial architecture, tend to force land values upward. This process not only yields taxable capital gains to individual landowners, including farmers who own a large proportion of the available land. It also raises basic costs to new users. Here is a pervasive, persistent factor making for price increases, to which the rise in our standards of living also contributes.

9. Water and some other natural resources in limited supply also tend to become scarcer in an economic sense, particularly as changing techniques and higher consumption standards make for increased per capita requirements. These operate to raise capital and product costs, though the aggregate effect on the price level may well be less than through rising land values. The resulting problems should not be minimized, but I can see no cause for alarm over the exhaustion of exhaustible resources in the near future.

10. Expansion of demands for food, fiber, and tobacco at rates assured by growth in adult-male equivalents threatens no shortages and is surely favorable to farmers. But it by no means assures a solution of the farm surplus problem, which continues to be aggravated by technological progress in farming and obsolete political programs of price support. The persistent

[33] Held, Harry (vice president, The Bowery Savings Bank): Adequate Housing for the Expected Increase in Population and Family Formation. In *ibid.*, II, 219–25. For the latest official projections of households and families by type, see *Current Population Reports*, Series P-20, No. 103, July 6, 1960.

[34] Hoffman, Morton: Economic Implications of Increasing Urbanization. . . . In *ibid.*, II, 233–41, and Sheppard Victor H.: The Financing of Municipal Governments. *In* PROBLEMS OF ECONOMIC DEVELOPMENT, *op. cit.*, II, 267–72. Hoffman says: "By 1977 the economic well-being of metropolitan areas may be the country's foremost economic concern."

decline in the farm population, and increasing proportions of their income derived from nonfarm sources, contribute only moderately to raise their per capita income.

IV. Pertinent Observations

1. Our vigorous population increase since 1940 has certainly contributed to our economic growth and freedom from severe postwar depressions. Similarly, the population prospects for the 1960s are favorable for both economic stability and more rapid economic growth, as well as for meeting the Communist threat, though they insure neither continuous prosperity nor "a 20-year boom." Keynes, Hansen, and others in the 1930s stressed the adverse effects on investment and national income from the retardation of population growth and the threatened decline in Western populations, and also their important influence in intensifying and prolonging business depressions.[35] Their reasoning was broadly sound, though their view of the population outlook soon proved wrong. Among other things Keynes well said:

> An increasing population has a very important influence on the demand for capital. Not only does the demand for capital —apart from technical changes and an improved standard of life—increase more or less in proportion to population. But, business expectations being based much more on present than on prospective demand, an era of increasing population tends to promote optimism, since demand will in general tend to exceed, rather than fall short of, what was hoped for. Moreover a mistake, resulting in a particular type of capital being in temporary over-supply, is in such conditions rapidly corrected. . . .

2. The Gross National Product reached a $500 billion rate in the first quarter of 1960, and the 1970 figure is now commonly

[35] Keynes, J. M.: Some Economic Consequences of a Declining Population. *Eugenics Review*, Apr. 1937, XXXIX, 13–17; Hansen, Alvin H.: Economic Progress and Declining Population Growth. *American Economic Review*, March, 1939, XXIX, 1–15; and Achinstein, Asher: Introduction to Business Cycles. Crowell, New York, 1950, pp. 373–87.

forecast at $750–800 billion in 1959 dollars.[36] Of the anticipated 50–60 per cent increase, a substantial fraction will be attributable to factors associated with our growth in population, families, and the labor force. If we succeed in better realizing our potentials, such forecasts will prove conservative.[37]

3. The decade ahead is surely rich in opportunities but it bristles with challenging problems, many of which grow out of population developments. These developments threaten increases in such evils as air and water pollution, noise, and congestion in various forms, and increased social costs to mitigate or eliminate these. For this decade at least, however, I cannot endorse Spengler's arguments that "population threatens prosperity" and that "undue population growth [*sic*] is currently tending to debase aesthetic values and to be fostered by such debasement."[38]

4. The need and opportunity for adult education, in the middle and older years of life, are rapidly growing as older knowledge becomes increasingly obsolete, as increased longevity and leisure permit more individuals to enlarge and modernize theirs, as married women take jobs after release from major household responsibilities, and as older persons seek employment after being retired from jobs or positions they have long held.[39] The prospective shortages in the teaching and medical professions, among others, call for increasingly effective development and utilization of human potentials for supplementing the skills of the great body of these professionals, and for evolving new

[36] United States Dept. of Labor: MANPOWER: CHALLENGE OF THE 1960s. (Washington, 1960), pp. 2–3; and National Industrial Conference Board: ECONOMIC GROWTH IN THE SIXTIES: PREREQUISITES, POTENTIALS, PROBLEMS. New York, May 1960.

[37] Knowles, James W.: THE POTENTIAL ECONOMIC GROWTH IN THE UNITED STATES. . . . (Joint Economic Committee print, Jan. 30, 1960).

[38] Spengler, J. J.: Population Threatens Prosperity. *Harvard Business Review*, Jan.–Feb. 1956, XXXIV, 85–94, and The Aesthetics of Population. *Population Bulletin* (Population Reference Bureau, Inc., Washington, D. C.), June 1957, XIII, 61–75. For a forceful presentation of a different view, see Turck, Fenton B.: The American Explosion, *Scientific Monthly*, 1952, LXXV, 187–91.

[39] See Stewart, Charles T. Jr.: Adult Education: America's Leading Economic Problem of the Next Twenty Years. In PROBLEMS OF ECONOMIC DEVELOPMENT, *op. cit.* II, 355–67.

types of jobs and new techniques to meet the swelling demands.

5. The prospective enlargement of the older population (projected at 24 per cent in the 1960s) deservedly attracts attention. Yet the notion that our "senior citizens" necessarily impose an increasingly heavy burden on the Nation's economy is ill-founded. To an extent seldom realized, older people are increasingly self-supporting despite low money incomes, if old age insurance benefits, pensions, self-service, mutual service, use of an owned home, and drafts on savings are all taken into account. Most of their needs and wants are simple and small. Increasingly, they are provided with prepaid medical, surgical, and hospital coverage at a cost within their own means or those of their children or interested relatives. There are of course gaps to be filled, as the current drive for expansion in such coverage at public expense testifies. Fuller utilization of talents and experience of oldsters, not only in unpaid activities but also in remunerative work if they want it, looms large among nationally justified objectives. Whatever net burden the elderly and aged really entail on the economy, moreover, will be the more easily borne as the Nation as a whole grows younger and increasingly productive.

6. The resumption of vigorous population growth since 1940, and its maintenance for two decades, reflect the vitality of the American people, the strength of non-material wants, strong preferences for marriage and family life, and renewed faith in America's future, as well as the generally high level of economic activity. The unexpected upsurge cannot be attributed to policies deliberately designed to promote population growth, though certain public measures incidentally contributed to it. Children have a higher place in American standards of living proper (i.e., levels desired with sufficient urgency to lead to sustained efforts to attain, maintain, or regain them)[40] than before World War II; and in the competition among more goods, more

[40] See my Standards and Content of Living. *American Economic Review,* Mar. 1945, XXXV, 1–15, and UN: REPORT ON INTERNATIONAL DEFINITION AND MEASUREMENT OF STANDARDS AND LEVELS OF LIVING. (United Nations, Mar. 1954, E/CN. 3/179, E/CN. 4/299), esp. pp. iii–vii, 5, 87–88.

leisure, earlier marriage, and more children, gains in leisure have been least.

7. The sorry experience of the ablest demographic, economic, and educational specialists in looking a decade ahead to the 1940s and 1950s should warn us not to be too sure of our ground as we look to the 1960s. Papers in this area are peculiarly subject to obsolescence, and one must reserve the right to change his views, without undue delay, as new evidence comes to light.

8. I have had to resist the temptation to examine our population prospects in the 1970s and beyond. Both tasks should be seriously undertaken, but no simple extrapolation can be trusted. If our postwar average rate of increase (over 1.7 per cent per year) cannot continue indefinitely, the timing and course of its eventual decline are not safely predictable. Whether the demographic developments in progress are wholesome or ominous for the longer future,[41] I have not discussed. Let me merely add that Americans are accustomed to rise to challenges, and that our economic and social history has typically confounded both superoptimists and pessimists of all degrees.

[41] For Philip M. Hauser's alarmist projective analysis, in an address of May 19, 1960, see *Population Bulletin* (Population Reference Bureau, Inc., Washington, D. C.), August 1960, XVI, 91– 106.

LIFE-TABLE FUNCTIONS FOR EGYPT BASED ON MODEL LIFE-TABLES AND QUASI-STABLE POPULATION THEORY

S. H. Abdel-Aty[1]

Introduction

THE census reports on age are usually highly defective in a majority of the underdeveloped countries. Corrections and adjustments frequently are necessary to get a distribution representing as nearly as possible the real age structure of the population.

As pointed out by Lotka, a population closed to migration and influenced by a relatively constant age schedules of fertility and mortality will attain, after a considerable number of years, a fixed rate of growth and a fixed age distribution and the population is said to be "stable."

Improvements in public health during the last decades, have caused a marked decline of mortality in many countries. A population with a constant fertility and a smoothly declining death rate may be termed a "quasi-stable" population, the age distribution at any moment is nearly identical to the stable with the given constant fertility and the current mortality schedule. This is almost the case in many of the underdeveloped countries at the present time.

The purpose of this paper is to utilize the properties of the quasi-stable population to derive a life table and the related vital rates for Egypt, based on the reported age distribution of the last census and the rate of natural growth in the last intercensal period. There is no need for correcting and smoothing the reported age distribution that we have to start with. The smoothed age distribution will follow as a result of the life table constructed.

The Model Life-Tables of the United Nations 1955, (10), make it possible to derive the survival rates at the different age groups for Egypt's population consistent with the average levels of human mortality as observed in the various countries of the world.

Survival rates and life expectancies so obtained are compared with the similar results given previously by other techniques. The com-

[1] Statistical Department, Ministry of Education, Egypt, U.A.R. This study was made while the author was at the Office of Population Research, Princeton University during the academic year 1959–1960. A letter received from the Author, February 7, 1961, indicated that he had recently moved to Tripoli to join the teaching staff of the Faculty of Science, Libya University.

parison shows that the previous life tables gave very high estimates for the survival rates and life expectancies to the old age-groups. It is then shown that these high survival rates were higher than in other countries known to have the highest life expectancy in the world.

The related points of the stable population theory and the effect of changes of fertility and mortality on the age structure of the population are reviewed to the extent required for explaining the calculation of the life table functions.

The Stable Population

For the human population, Lotka (6) has shown that with constant age schedules of mortality and fertility, the population will ultimately have a constant rate of growth and a fixed age distribution. The proportion having age a in the stable population is given by

$$C(a) = be^{-ra}p(a) \qquad [1]$$

where

$C(a)$ = the density of age distribution such that, out of a total number N of persons, a number of $NC(a)da$ are comprised within the age limits a and $a + da$

b = birth rate per head per annum

r = the rate of natural increase or the excess of birth rate b over death rate d i.e., $r = b - d$

and $p(a)$ = the probability of surviving a years after birth which equals the life table number of survivors l_a at age a divided by the size of the cohort at birth l_o.

Usually, the age distribution is given by the proportion in an age interval a to $a + n$ say, and therefore

$$C(a, a + n) = \int_a^{a+n} be^{-ra}\frac{l_a}{l_o}da \qquad [2]$$
$$\doteq be^{-r(a+\frac{1}{2}n)}{}_nL_a/l_o$$

The proportions in all the age groups will add up to 1. Therefore

$$1 = \int_0^{\infty} be^{-ra}\frac{l_a}{l_o}da$$

from which the birth rate of the stable population is

$$b = l_o / \int_0^w e^{-ra} l_a da \qquad [3]$$

$$= l_o / \sum_{a=0}^{w} e^{-r(a+\frac{1}{2}n)} {}_nL_a$$

and consequently, the proportion of the population in the age interval a to a + n is given by

$$C(a, a+n) = e^{-r(a+\frac{1}{2}n)} {}_nL_a / \sum_{a=0}^{w} e^{-r(a+\frac{1}{2}n)} {}_nL_a \qquad [4]$$

where

${}_nL_a$ = the life table proportion in a given age group

and l_o = the radix of the life table.

The second fundamental equation for the stable population is that the birth rate

$b = \int C(a)m(a)da$, limits of integration over the fertility age range

which by substituting for C(a) becomes

$$1 = \int_0^w e^{-ra} p(a)m(a)da \qquad [5]$$

where

m(a) = the maternity frequency per annum of females of age a.

This equation is an implicit expression for the stable rate of growth r. Solving for the real root of r satisfying this equation, Dublin and Lotka (6) found that r is the root of the quadratic equation $\frac{1}{2}\beta r^2 + \alpha r - \log_e R_o = o$ given by

$$r = \frac{1}{\beta}\{-\alpha + \sqrt{\alpha^2 + 2\beta \log_e R_o}\} \qquad [6]$$

where

$$\alpha = R_1/R_o \qquad \text{and} \qquad \beta = \left(\frac{R_1}{R_o}\right)^2 - \frac{R_2}{R_o}$$

and R_o, R_1, R_2 are given by

$$R_n = \int_0^w a^n p(a)m(a)da \qquad \text{for } n = o, 1, 2.$$

Effect of Mortality Change on the Stable Age Distribution

Coale (3, 4) has shown that the decline of mortality—with a higher life expectancy—has a relatively minor effect on the stable age distribution provided that levels of fertility are unchanged. A more favorable mortality schedule—with a higher life expectancy—will yield a stable population with: (a) a higher proportion under 15, (b) a distribution with a lower average age, and (c) a slight increase in the fractions of the ages over 60, *see* Figure 1 for illustration and Figures 8 and 9 for Egypt's population.

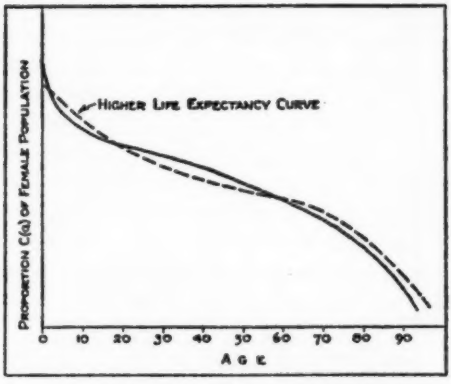


Fig. 1. Hypothetical curves indicating the effect of mortality change on the stable age distribution.

Now let us consider a population in which fertility remains constant and mortality declines. This is the case in a majority of the populations of underdeveloped countries at the present time. Let us imagine that at a given moment mortality ceases to decline and remains constant at the level attained in that moment. After a certain time the population will attain the stable population structure. However, at that moment, as pointed out by Bourgeois-Pichat (2), the population is already very close to this stable form. It follows that the population in which fertility remains constant and mortality declines, may be considered similar at each moment to a stable population. In other words, in these populations, there is at each moment between the age composition, the birth rate, the mortality rate and the rate of increase approximately the same relations as in a stable population.

Effect of Fertility Change on the Stable Age Distribution

The role of fertility in shaping the stable age distribution is much more effective than that of mortality. This is known at least within the prevailing levels of mortality and fertility observed in the World.

Differences in fertility produce quite different stable age distributions. This, combined with the fact that changes in mortality only slightly affect the age structure, means that a schedule of fertility by age is sufficient to give a fair approximation to the stable age distribution even if mortality rates by age are not known.

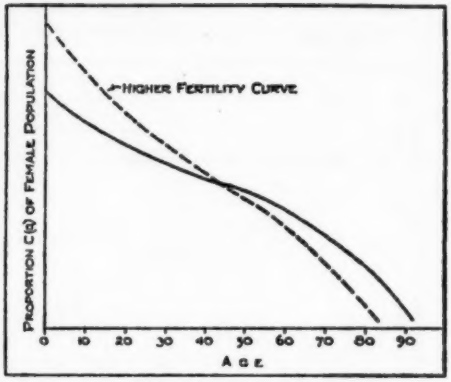


Fig. 2. Hypothetical curves indicating effect of fertility change on the stable age distribution.

If a high fertility age distribution is compared with a low fertility distribution with the same mortality, the higher fertility population will have higher proportions at younger ages, the two distributions will tend to intersect at the mean of the average ages, Figure 2.

The heavy influence of fertility on the stable age distribution is due to the fact that the two terms b and $\int_{15}^{49} C(a)da$ which define the general fertility rate (the general fertility rate = no. of live births/no. of women aged 15–49) are respectively the ordinate of the age proportion distribution curve at its origin, given by $C(o) = b$, and the area under the middle part of the curve, given by $\int_{15}^{49} C(a)da$. Thus if the general fertility rate is increased, the starting point of the age curve will rise by almost the same proportion but the change in the middle area of the curve will be comparatively small. This means that we shall have higher proportions at the younger ages. In general the approximate form of the age distribution is determined by the level of fertility. The level of mortality has more or less second order effects on the distribution.

Patterns of Mortality and Model Life-Tables

The typical variation of mortality with advance in age during the life span is represented by a "u" shaped curve which starts high at birth, declines rapidly to a minimum around age 12, and then increases

slowly through adolescence and maturity and finally rises sharply at old ages.

Improvements in conditions of living and standards of health are reflected in the improvements in mortality rates which change from high to low levels. Even at the generally low levels of mortality the variations by age are still characterized by the "u" shaped pattern.

The patterns of transition from high to low mortality levels during the last fifty years have been studied by the Population Branch of the Bureau of Social Affairs of the United Nations (10), Series A/22. The study covered 158 selected national life tables of 50 different countries of all the continents. For every age group there was a maximum and a minimum value of all the different specific mortality rates at that age. The ratio between maximum and minimum mortalities becomes smaller and smaller as we pass from younger to older age-groups. That is, the variations between mortality rates in the World are less discernible in the older ages whereas younger ages are more sensitive to changes in mortality levels. Consequently, an appropriate measure of the general transition from high to low mortality must be sought in the lower age brackets, where the variations of mortality are relatively large.

For every pair of consecutive age-groups, the scatter points of the world mortality rates ${}_5q_x$ and ${}_5q_{x+5}$ of the two age-groups are plotted and a second-degree parabola of the form

$$ {}_5q_{x+5} = A + B\,{}_5q_x + C\,{}_5q_x^2 \qquad \text{for } x = 0, (5), 75 $$

is fitted. Also a similar parabola is fitted to q_0 and ${}_5q_0$. Therefore it will be sufficient to know the level of infant mortality q_0 (or the level of mortality at a young age-group), then all of the mortality rates at the different age groups are estimated from these equations. The estimates are at best possible averages of the World's mortality rates. For forty equally spaced levels of infant mortality q_0, the Model Life-Tables mortality rates ${}_5q_x$ (for $x = 0, (5), 80$) were given in Series A/22, (10). The other functions of the Model Life-Tables were tabulated in Series A/25, (11).

The Growth of Population in Egypt

According to the 1897 census, Egypt's population was then 9.734 millions. Decennial censuses afterwards show a gradual increase in the population. The census of 1947 indicated a population of 18.967 millions. That is, the population has nearly doubled in 50 years. It

is found that there was overcounting in the 1947 census. An adjustment for overenumeration given by El-Badry (7) reduced it to 17.907 millions in 1947. In plotting the population size in the different census years, Table 1 and Figure 3, we see that the population growth since 1897 is almost constant as the census points form a line which is nearly straight. The 1947 returns, however, show a higher rate of increase than that prevailing in the previous censuses and the adjustment for overenumeration was made by the continuation of the 1907–1937 rate of increase. In 1957, the preenumeration stages were conducted, but because of the unfavorable conditions that occurred in that year the census was postponed. It was conducted in 1960 for the new United Arab Republic, the north and the south regions (Syria and Egypt). According to estimates from the National Sample Survey of Labor Force in Egypt, the 1957 population was 23.632 millions. This estimate shows a higher rate of population growth in the last decade than that during 1907–1937 or 1907–1947. Patterns of birth and death rates in Egypt during the last years will indicate, as shown later, an increase in the rate of growth during the last decade. The rates of growth r per head per annum during the

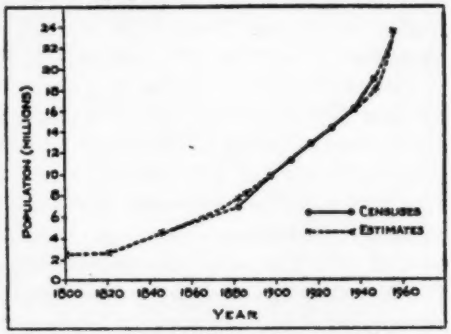


Fig. 3. Population of Eygpt, 1800 to 1957.

Table 1. Population of Egypt (in thousands).

Census Year	Males	Females	Total
1897	4,948	4,787	9,734
1907	5,617	5,573	11,190
1917	6,369	6,349	12,718
1927	7,058	7,120	14,178
1937	7,967	7,954	15,921
1947	9,392	9,575	18,967
1947[a]	8,951	8,956	17,907
1957[b]	11,789	11,843	23,632

[a] Estimation of El-Badry (7) for correcting overenumeration.
[b] Estimate from The National Labor Force Sample Survey.

intercensal period t years are calculated by the exponential law

$$r = \frac{1}{t} \log_e (P_t/P_o) \qquad [7]$$

$$= 2.302585 \times \frac{1}{t} \log (P_t/P_o)$$

where P_o is the population size at the start of the interval t and P_t is its size by the end of that interval.

Population Rate of Growth (r) per head per year, for Egypt

Decade	*Males*	*Females*	*Males and Females*
1897–1907	.012681	.015203	.013939
1907–1917	.012566	.013036	.012800
1917–1927	.010274	.011461	.010865
1927–1937	.012116	.011076	.011594
1937–1947	.011645	.011864	.011755

The tabulated rate of growth for the 1937–1947 decade is for the adjusted value of 1947 census. If the reported value of the 1947 census is considered, the rate of growth in 1937–1947 is .017505 (for both sexes). Also, in the decade 1947–1957, the rate is found to be .021989 (for both sexes). Thus during the last one or two decades, it appears that the rate of growth has been rising above the rate that had remained almost the same between 1897 and 1947, when it was between .011 and .014 per head per year. From 1947 until 1960 no full census was taken. It is hoped that the 1960 census will provide a more reliable estimate of growth rate in the last decade.

Birth and Death Rates in Egypt

Birth and death rates exhibit serious underregistration in almost all the low-income countries. Despite the fact that in Egypt registration of births and deaths has been compulsory by law for quite a number of years, there is nevertheless a high percentage of underregistration. The underregistration of births is most acute in villages located far from the city or from the health bureau area. The decree for registering births, issued in 1912, imposes the responsibility for reporting on the mother, father, or any relative in the same household. It also imposes responsibility on the doctor or nurse who managed the delivery. Birth registration should be within fifteen days

after the birth day. For deaths, notification is the responsibility of relatives or any member of the household. A death should be reported by the doctor or health representative or by the "Omda" or "Sheikh-el-balad" who are representatives of the area. Registrations of births and deaths usually are made in the public health offices. In remote areas registration is made in the tax collector's office (called Sarraf). The public health offices send a weekly report of these events to the Statistical Department; the tax collectors' offices send their reports monthly. It is essential for every person in the country to submit a birth certificate for employment, civil service, identification, army service and compulsory school enrollment. Persons that escaped birth registration will need to register themselves on entering school or employment.

An estimate of the percentage of underregistration of births based on a comparison of the crude birth rates in the health bureau areas with that in the whole country is found to be about 4 per cent during

Table 2. Crude birth and death rates per 1,000 of population in Egypt.

A—Average Rates in 5 Year Periods, from 1906 to 1954

Period	All Areas		Health Bureau Areas	
	Birth Rate (Average)	Death Rate (Average)	Birth Rate (Average)	Death Rate (Average)
1906–1909	43.0	25.5	44.2	
1910–1914	42.5	26.2	45.2	
1915–1919	39.8	31.3	40.8	41.0
1920–1924	42.8	25.8	48.7	32.6
1925–1929	43.9	26.5	45.8	32.8
1930–1934	43.7	27.0	45.2	28.8
1935–1939	42.8	26.9	45.0	29.6
1940–1944	39.6	26.8		
1945–1949	42.4	23.0		
1950–1954	43.9	18.7		

B—Single Year Rates, from 1935 to 1955

Year	Birth Rate	Death Rate	Year	Birth Rate	Death Rate	Year	Birth Rate	Death Rate
1935	41.3	26.4	1942	37.6	28.3	1949	41.8	20.6
1936	44.2	28.8	1943	38.7	27.7	1950	44.4	19.1
1937	43.4	27.1	1944	39.8	26.0	1951	44.8	19.3
1938	43.2	26.3	1945	42.7	27.7	1952	45.1	17.7
1939	42.0	25.9	1946	41.2	25.0	1953	42.5	19.5
1940	41.3	26.3	1947	43.8	21.4	1954	42.5	17.8
1941	40.4	25.7	1948	42.7	20.4	1955	40.0	17.6

the period 1925 to 1940. Underregistration can also be estimated by projecting the number of children under 5 years of age in the census to births by the division ${}_5L_0/{}_5l_0$ (the probability of surviving from birth to ages zero to 5) and comparing the projected result with the registered number of births in the five years before the census. This method will give, using the 1947 census data, an estimate of about 7 per cent for birth underregistration.

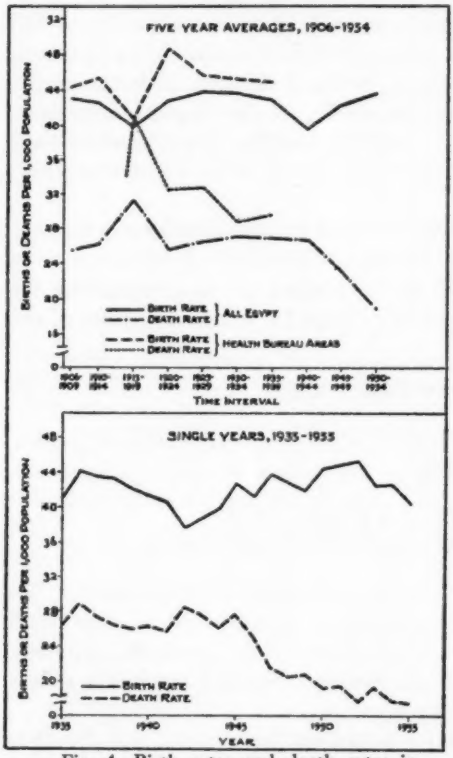


Fig. 4. Birth rates and death rates in Egypt (five year averages), 1906–1954.

Variations of birth rates in Egypt were not wide over a long period of time. As seen in Table 2 and Figure 4, the registered crude birth rate averages between 42 and 44 births per thousand of population. The nearly fixed rate extends from 1906 to 1954, with the exception of the two periods 1915–1919 and 1940–1944 which reflect the effects of the World Wars I and II. The annual variations, as seen in the last twenty years, never diverge from this fixed birth rate by more than 2 per thousand.

The annual death rate during the twenty years preceding 1945 remained almost constant up to 1945, according to the reported figures. (These figures were considerably underreported, especially in the earlier years, as will be seen later.) The average death rate from 1935 to 1945 was 27 per thousand of population. The annual variations from this average do not exceed 2 per thousand. After

1945 a clear decline of death rate took place at the rate of about one per thousand per annum.

The Census Age Distribution

Census age reports in Egypt are, as in many other countries, full of errors. Heapings at ages ending in 0, 5, and some even numbers, overreporting of old ages, underreporting of children, underestimation of females in certain age groups as 40–49, etc., are typical distortions. Other types of errors are quite frequent, and the data frequently have been adjusted and smoothed before being used for demographic analysis.

Different methods have been followed for age-distribution smoothing. In most cases smoothing by fitting a frequency distribution curve was followed. Kiser (9) and El-Shanawany (8) in constructing life tables for Egypt fitted the Pearson Type IX frequency curve of the form

$$y = y_o(a - x)^m \qquad [8]$$

to the age distributions. For an Indian life-table a Type I frequency curve was applied of the form

$$y = y_o\left(1 + \frac{x}{a_1}\right)^{m_1}\left(1 - \frac{x}{a_2}\right)^{m_2} \qquad [9]$$

where the age span w is $a_1 + a_2$, and the origin is at the mode.

Before smoothing, adjustments of the irregularities in some parts of the age distribution are often made. These methods, although clearly improving the raw data, are laborious and inevitably reflect the personal judgment of the author.

The method applied here for adjusting the reported age distribution is based on the following characteristics of the population:

1. Fertility can be considered as practically constant.
2. Mortality has varied in recent years smoothly, and in accordance with the typical age-patterns of mortality prevailing in the world.
3. Absence or negligible importance of international migration.

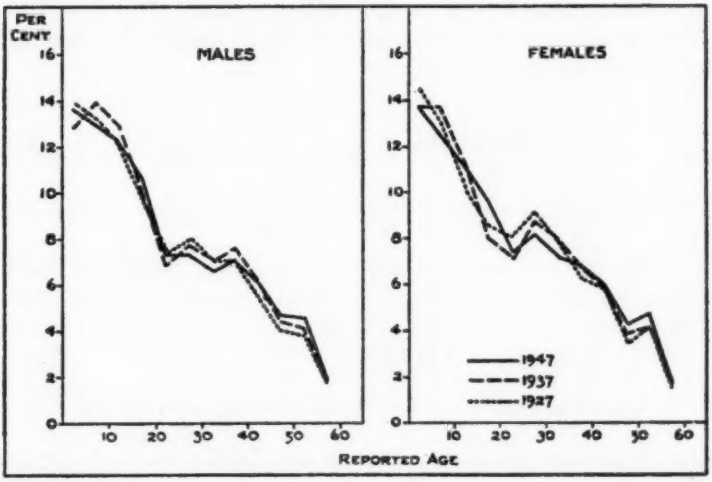
These basic conditions already characterize the population of Egypt as well as many of the other low-income countries. Unchanging fertility and fixed mortality will lead to the stability of the population with ultimately fixed age structure. Changes in mortality will introduce only slight changes in the stable age structure and the population

	Males			Females		
	1947	1937	1927	1947	1937	1927
0– 4	13.6	13.0	14.1	13.6	13.7	14.5
5– 9	12.9	13.9	13.3	12.4	13.8	12.9
10–14	12.2	12.9	12.2	11.2	11.0	10.1
15–19	10.5	8.9	9.6	9.6	8.0	8.6
20–24	7.2	6.8	7.4	7.4	7.1	8.1
25–29	7.3	7.7	8.1	8.2	8.7	9.2
30–34	6.6	7.0	7.0	7.2	8.0	8.0
35–39	7.0	7.5	7.0	6.8	6.8	6.2
40–44	6.1	6.0	5.4	5.9	5.9	5.8
45–49	4.6	4.3	4.0	4.3	3.9	3.4
50–54	4.5	4.1	3.8	4.7	4.2	4.3
55–59	1.8	1.8	1.7	1.8	1.7	1.5
60+	5.5	5.8	6.1	6.5	6.9	7.0
Not Given	0.3	0.2	0.3	0.3	0.2	0.3
Total	100.1	99.9	100.0	99.9	99.9	99.9

Table 3. Reported age distribution (in percentages of total population) in the census years 1927, 1937 and 1947.

will be approximately stable, i.e. "quasi-stable." The similarity of the reported age distributions of the 1927, 1937 and 1947 censuses is seen in Table 3 and Figure 5.

Fig. 5. Percentage age distributions, by sex, as reported in the censuses of Egypt 1927–1947.



Finding the Life Table Functions

The reported age distribution of the census of 1947 and the rate of growth (r) in the period between the last two censuses

(r = .011645 for males and r = .011864 for females)

are assumed as characteristics of a "quasi-stable" population.

Recalling the first fundamental formula of stable population, we get

$$C(a)e^{ra} = bl_a/l_o \qquad [10]$$

If $\frac{n(a)}{N}$ is the reported proportion of population in the age-group whose mid-point is a, then $\frac{n(a)}{N} e^{ra}$ will be proportional to l_a. The values of l_a so obtained from the census data will be subject to all the irregularities and errors of age reporting; we shall call it "l_a reported" to distinguish it from the life table l_a which represents the survivors at age a.

An average l_a value covering all ages or at least from age five or ten and over (in which case we avoid the usually most erroneous group of under five years of age) will be fairly reliable. The errors of age reporting will to a large degree neutralize each other as the heapings in certain ages are usually at the expense of the neighboring ages. The person-years in the life table above age 10 (T_{10}) as calculated from the "l_a reported" values will approximate fairly well the value that would be calculated from an actual life table.

Now, to estimate the expectation of life at age ten, $\mathring{e}_{10}$, from the "l_a reported" values, we have:

$$\text{estimate of } \mathring{e}_{10} = \frac{1}{l_{10}} \int_{10}^{\infty} l_x dx = \frac{T_{10}}{l_{10}} \qquad [11]$$

where the l's are the reported values given by the census age distribution.

To evaluate T_{10}, the average between two successive reported l_x values is multiplied by the length of the age group and then summed for all age groups from 10 and over. For the last age group, 85 years and over, approximation is given by

$$L_{85+} = l_{85} \log_{10} l_{85} \qquad \text{(From Manual III, U.N. p. 23).}$$

More specifically, when the reported age distribution is in 5-years

age-groups, we shall have the set of values of reported $l_{2.5}$, $l_{7.5}$, . . . , $l_{82.5}$ and $l_{87.5}$. Therefore

$$l_{10} = \frac{1}{2}(l_{7.5} + l_{12.5}) \qquad [12]$$

and

$$T_{10} = 5[.25(l_{7.5} - l_{12.5}) + \sum_{a=10}^{80} l_{a+2.5} - .25(l_{82.5} - l_{87.5})] + L_{85+} \qquad [13]$$

The estimates of expectation of life at age 10, from the 1947 data, give the following results (see Table 4):

for males: estimate of $\mathring{e}_{10} = 38.732$ years

for females: estimate of $\mathring{e}_{10} = 42.006$ years

With these estimates of $\mathring{e}_{10}$, we get the life table function l_x, which

Table 4. Reported age proportions $\frac{n(a)}{N}$ and reported survivors (l_a reported) for the 1947 Census.

Pivotal Age (a)	Males			Females		
	$\frac{n(a)}{N}$	$e^{.0118(a)}$	l_a	$\frac{n(a)}{N}$	$e^{.0118(a)}$	l_a
	(1)	(2)	(1) × (2)	(1)	(2)	(1) × (2)
2.5	.1362	1.0295	.1403	.1363	1.0301	.1404
7.5	.1287	1.0913	.1405	.1244	1.0931	.1360
12.5	.1216	1.1567	.1407	.1119	1.1599	.1298
17.5	.1048	1.2260	.1285	.0958	1.2307	.1179
22.5	.0722	1.2995	.0938	.0737	1.3060	.0963
27.5	.0730	1.3775	.1006	.0821	1.3858	.1138
32.5	.0660	1.4600	.0964	.0720	1.4705	.1059
37.5	.0702	1.5476	.1086	.0683	1.5603	.1065
42.5	.0606	1.6404	.0994	.0591	1.6557	.0979
47.5	.0456	1.7387	.0793	.0434	1.7569	.0762
52.5	.0449	1.8429	.0827	.0468	1.8643	.0873
57.5	.0182	1.9534	.0356	.0181	1.9782	.0358
62.5	.0268	2.0706	.0556	.0312	2.0991	.0655
67.5	.0089	2.1947	.0196	.0086	2.2274	.0191
72.5	.0115	2.3263	.0267	.0143	2.3635	.0338
77.5	.0025	2.4657	.0062	.0025	2.5079	.0063
82.5	.0037	2.6136	.0097	.0055	2.6612	.0146
87.5	.0018	2.7703	.0050	.0025	2.8239	.0070
	Estimate of $\mathring{e}_{10} = 38.7$			Estimate of $\mathring{e}_{10} = 42.0$		

is the survivors to age x out of a cohort of 100,000 at birth, by interpolation from the Model Life Tables in Manual III U.N. Series A/25, (11). Before interpolating, we must get the tabular values of $\mathring{e}_{10}$ between which our estimates lie. These are given by

$$\mathring{e}_{10} = \frac{1}{l_{10}} (T_0 - {}_{10}L_0) \qquad [14]$$

where l_{10}, T_0 and ${}_{10}L_0$ are Model Life-Table values.

The numerical tabular values of $\mathring{e}_{10}$ corresponding to the tabular levels of $\mathring{e}_0$ are as follows

level of $\mathring{e}_0$	Model Life-Table values of $\mathring{e}_{10}$	
	M	F
27.5	27.4816	37.3924
30.0	39.1012	39.2709
32.5	40.7370	41.1702
35.0	42.4373	42.9376

The other functions are calculated by the following simple relations:

(i) The survivors in the age group x to x + 5 are derived by interpolation from the ${}_5L_x$ table or by calculation from

$$\left.\begin{aligned} {}_5L_x &= 2.5(l_x + l_{x+5}) && \text{for age groups 5–9 to 80–84} \\ L_0 &= .25l_0 + .75l_1 && \text{for the first year of life} \\ {}_4L_1 &= 1.9l_1 + 2.1l_5 && \text{for age group 1–4} \\ {}_5L_0 &= .25l_0 + 2.65l_1 + 2.1l_5 && \text{for age group 0–4} \end{aligned}\right\} \qquad [15]$$

$$\left.\begin{aligned} \text{(ii)}\quad {}_5q_x &= \frac{1}{l_x}(l_x - l_{x+5}) = \text{probability of dying between age x and x + 5} \\ {}_5p_x &= 1 - {}_5q_x = \text{probability of surviving from age x to age x + 5} \end{aligned}\right\} \qquad [16]$$

$$\left.\begin{aligned} \text{(iii)}\quad T_x &= \sum_x^w {}_5L_x = \text{total number of years of life remaining to survivors at age x} \\ \text{and } \mathring{e}_x &= \frac{T_x}{l_x} = \text{average number of years remaining to survivors at age x} \end{aligned}\right\} \qquad [17]$$

and thus the life table functions are calculated as given in Table 5.

Age x	Males			Females		
	l_x	${}_1p_x$	$\mathring{e}_x$	l_x	${}_1p_x$	$\mathring{e}_x$
0	100,000	.6155	29.0	100,000	.6784	34.3
5	61,561	.9450	41.5	67,834	.9538	45.0
10	58,174	.9646	38.7	64.701	.9670	42.0
15	56,105	.9514	35.1	62,563	.9541	38.4
20	53,379	.9351	31.8	59,690	.9396	35.1
25	49,913	.9276	28.8	56,082	.9315	32.2
30	46,297	.9179	25.8	52,239	.9253	29.4
35	42,494	.9035	22.9	48,335	.9187	26.6
40	38,395	.8817	20.1	44,404	.9119	23.7
45	33,851	.8547	17.5	40,493	.8986	20.7
50	28,932	.8239	15.0	36,387	.8770	17.8
55	23,837	.7820	12.7	31,910	.8458	14.9
60	18,641	.7294	10.5	26,988	.7931	12.2
65	13,597	.6540	8.5	21,403	.7200	9.7
70	8,893	.5440	6.7	15,411	.6128	7.5
75	4,838	.4161	5.1	9,444	.4758	5.7
80	2,013	.2573	3.8	4,493	.3181	4.3
85	518		2.7	1,429		3.2

Table 5. Life table for Egypt in the period 1937–1947.

The Adjusted Age Distribution

Unlike the usual procedures of correcting the census reported data and then constructing the life table, the process here is reversed by constructing the life table first and from the life table functions the adjusted age distribution is derived. The proportion of the population in the age group a to a + n is, on the basis of stable age distribution, given by equation [4].

$$C(a, a + n) = e^{-r(a+\frac{1}{2}n)}{}_nL_a / \sum_{a=0}^{w} e^{-r(a+\frac{1}{2}n)}{}_nL_a$$

This procedure was followed in calculating the proportional age distribution in Table 6. Figures 6 and 7 show how adequately this procedure gives smoothed age distributions fitting the reported distributions of the census and correcting its errors.

Intrinsic Vital Rates

The birth rate associated with the quasi-stable population is given by equation [3]

$$b = l_0 / \sum_{a=0}^{w} e^{-r(a+\frac{1}{2}n)}{}_nL_a$$

Pivotal Age (a)	Males $e^{-.011604a}$ (1)	Males $_1L_a/l_0$ (2)	Males (1) × (2)	Males C(a)	Females $e^{-.011604a}$ (1)	Females $_1L_a/l_0$ (2)	Females (1) × (2)	Females C(a)
2.5	.9713	3.5056	3.4050	.158	.9708	3.7660	3.6560	.148
7.5	.9164	2.9934	2.7431	.127	.9149	3.3134	3.0313	.122
12.5	.8645	2.8570	2.4700	.115	.8622	3.1816	2.7431	.111
17.5	.8156	2.7371	2.2325	.104	.8125	3.0563	2.4833	.100
22.5	.7695	2.5823	1.9871	.092	.7657	2.8943	2.2162	.089
27.5	.7260	2.4052	1.7461	.081	.7216	2.7080	1.9541	.079
32.5	.6849	2.2198	1.5203	.071	.6801	2.5143	1.7099	.069
37.5	.6462	2.0222	1.3067	.061	.6409	2.3185	1.4859	.060
42.5	.6096	1.8061	1.1011	.051	.6040	2.1224	1.2819	.052
47.5	.5751	1.5696	.9027	.042	.5692	1.9220	1.0940	.044
52.5	.5426	1.3192	.7158	.033	.5364	1.7074	.9159	.037
57.5	.5119	1.0620	.5436	.025	.5055	1.4724	.7443	.030
62.5	.4830	.8059	.3892	.018	.4764	1.2098	.5763	.023
67.5	.4556	.5623	.2562	.012	.4490	.9203	.4132	.017
72.5	.4299	.3433	.1476	.007	.4231	.6214	.2629	.011
77.5	.4056	.1713	.0695	.003	.3987	.3484	.1389	.006
82.5	.3826	.0633	.0242	.001	.3758	.1480	.0556	.002
87.5	.3610	.0141	.0051	.000	.3541	.0451	.0160	.001
Totals			21.5658	1.000			24.7788	1.000
Intrinsic birth rate:			.0464		Intrinsic birth rate		.0404	
Intrinsic death rate:			.0347		Intrinsic death rate		.0285	

Table 6. Intrinsic birth and death rates and the stable age proportions, C(a), of Egypt's population in 1947.

The intrinsic birth rate for males (b_m) and for females (b_f) calculated by this formula are given in Table 6 and are as follows:

b_m = 46.370 boys born per 1000 of male population

b_f = 40.357 girls " " " " female "

For the combined birth rate of males and females we may estimate the sex ratio. During the five years 1945–1949, there were 4,040,255 live births of whom 2,116,696 were males, therefore,

$$\text{Sex ratio of females} = \frac{\text{Female births}}{\text{Total births}}$$

$$= .4761$$

Ratio of male births to female births is

$$m:f = .5239/.4761 = 1.1004:1$$

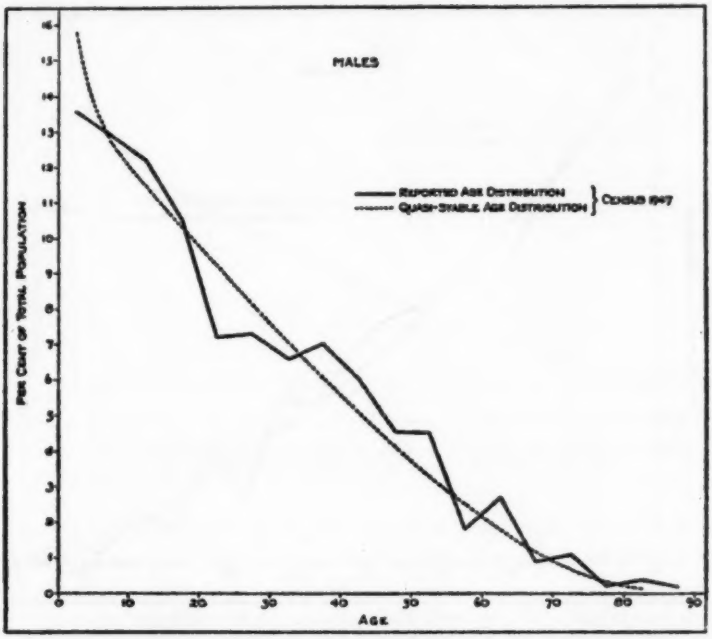


Fig. 6. Reported age distribution and quasi-stable age distribution of males in Egypt, Census 1947.

The combined birth rate b is the harmonic weighted average of the male and female birth rates and given by

$$b = (m + f)/\left(\frac{m}{b_m} + \frac{f}{b_f}\right) \qquad [18]$$

which is found to be of the value

$$b = 43.620 \text{ children born per 1000 of population.}$$

The corresponding intrinsic death rates as given by $d = b - r$ have the following values

$$d_m = 34.7 \text{ per 1000 of male population}$$
$$d_f = 28.5 \text{ " " " female "}$$
and $$d = 31.9 \text{ " " " population}$$

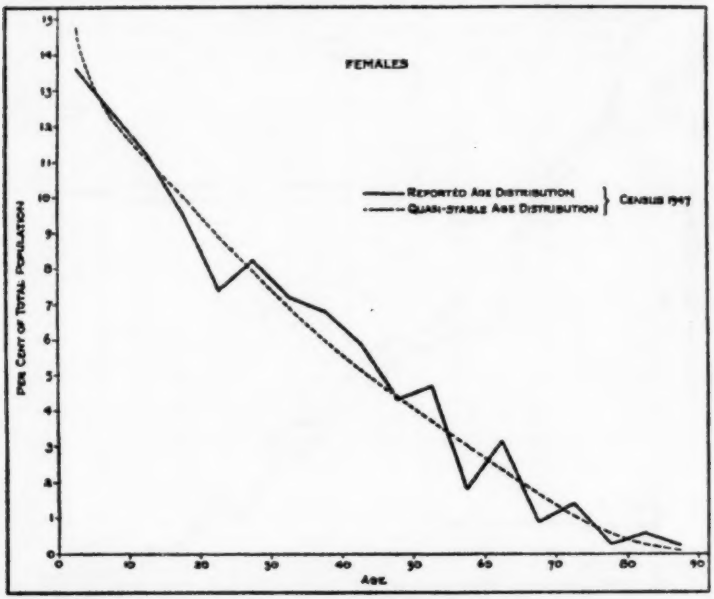


Fig. 7. Reported age distribution and quasi-stable age distribution of females in Egypt, Census 1947.

The intrinsic, or standardized, birth and death rates represent the ultimate, or long run average, of crude birth and death rates if the population remains subject to constant conditions of fertility and mortality. When this standardized situation prevails, the ultimate annual rate of growth (r) of the population will remain constant and the same for both sexes, and the age structure will be ultimately fixed. A comparison of the intrinsic rates with the crude rates between 1937 and 1947 is given in the following table

	Crude Rate per 1,000 Population in 1937–1946			Intrinsic Rate per 1,000 Population
	Maximum	Minimum	Average	
Births	43.4	37.6	41.0	43.6
Deaths	28.3	25.0	26.6	31.9

This comparison suggests that birth registration was fairly complete while death registration was far from complete. The underregistra-

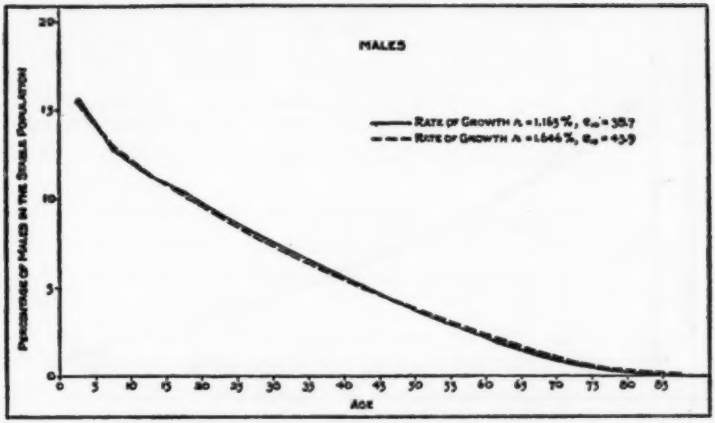


Fig. 8. Chart showing slight effect of improved mortality on age structure of stable population of Egyptian males. Basic data from 1947 Census.

tion of births was about 6 per cent of the average crude birth rate while the underregistration of deaths was 20 per cent of the average crude death rate.

It is interesting to ascertain what the intrinsic birth and death rates would be if the population of Egypt in 1947 were 18.967 millions as given by the census reports. Also, how would the estimated age structure of the population be affected if the official figures were accepted as correct rather than as inflated?

If the population in 1947 were 18.967 millions, the rate of increase during the decade 1937–47 would be .01646 for males, .01855 for females and .01751 for both sexes. Following the same procedure for constructing the life table and the stable age distribution, it is found that the expectation of life at age ten will be

$$\mathring{e}_{10} = 43.9 \text{ years for males}$$

and

$$\mathring{e}_{10} = 50.4 \text{ “ “ females}$$

The stable age distribution for these levels of $\mathring{e}_{10}$, which are much higher than the 38.7 and 42.0 found before, give a typical illustration of the effect of a decline in mortality on the stable age structure. Figures 8 and 9 show how slight are the changes in the age structure of Egypt's population when mortality declines.

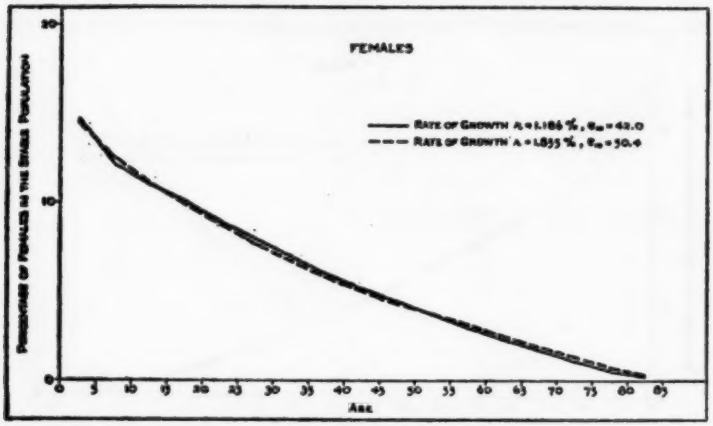


Fig. 9. Chart showing slight effect of improved mortality on age structure of stable population of Egyptian females. Basic data from 1947 Census.

The intrinsic vital rates are found to be:

b = 39.7 children born per 1,000 of population

and d = 22.2 deaths per 1,000 of population.

Comparing these with the crude rates will show that: (a) the assumed increase of the population implies a very substantially lower level of mortality and a slightly lower level of fertility; (b) the low value of the intrinsic death rate in comparison with the crude rate (which is probably too low because of incomplete death registration) supports the view that the 1947 census was a relative overenumeration.

Evaluation of Results

To judge the results given by the technique followed in this paper, the following comparisons were made:

1. Comparison of the intrinsic vital rates of birth and death with the actual reported rates.

2. Comparison of the stable age structure of the population with the reported age structure of the census.

3. Comparison of the life table functions of survival rates and life expectancies with those of life tables constructed by other methods.

Comparisons (1) and (2) have been covered in the previous articles of the text and proved adequacy of results were obtained. For com-

parison with the life tables constructed by previous authors, the following other life tables for Egypt were considered:

1. The "First National Life-Table for Egypt" for the period 1917–1927 constructed by El-Shanawany (8) in 1936. The method applied was based on smoothing the reported age distribution by a Pearson type IX curve and following cohorts in two censuses to get the survival rates.

2. Life-Table for the period 1927–1937 constructed by Kiser (9) in 1944 by adjusting[3] the census age distribution and smoothing by curve fitting. Similar in principle to the census differencing method used by El-Shanawany.

3. "The Egyptian National Life-Table No. 2" for the period 1936–1938 by Abdel-Rahman (1) in 1948 constructed by use of death reports and the census age distribution of the population to calculate the age-specific death rates.

4. Life-Table for the period 1907–1947 constructed by El-Badry (7) in 1955. The procedure was based on the stability of the census age distribution. According to the stable population theory, the proportion of the population aged a will be

$$C(a) = be^{-ra}l_a/l_o$$

where the terms have the same meanings as given before. From this equation the survival rate from age a to age a + n is deduced as follows

$$\frac{C(a+n)e^{r(a+n)}}{C(a)e^{ra}} = \frac{l_{a+n}}{l_a} = {}_nP_a$$

Therefore, to calculate the life table functions, it was necessary to correct the reported age distribution of the census to get an estimate of C(a). A series of corrections was made of the census age frequencies, as correction of female frequencies under 20 years of age, correction of male frequencies in the first three age-groups, correction for underestimation of females at ages 40–59, and correction for the old age frequencies. Having corrected the frequencies of the five censuses

[3] Kiser's adjustment to the census age-groups reports:

0– 4 No change except adjustment for underenumeration
5– 9 Census figure plus one-fourth of age 10
10–14 Census figure less one-fourth of age 10 plus one-half of age 15
15–19 " " " one-half " " 15 " " " " " " 20
20–24 " " " " " " " 20 " " " " " " 25
etc.

from 1907 to 1947 the average is taken as an estimate of the frequency C(a) at age a. Hence starting with the above equation of ${}_{n}p_{a}$ the life table was constructed.

It was necessary to give a brief hint on each of the methods used in order to reveal the reasons for any discrepancies.

The expectation of life $\mathring{e}_x$ for a given age x is plotted for the different life tables in Figure 10.

The following points are notable:

1. The lines form a close bundle, a situation which conforms with the fact of stabilization of the population structure over the whole period 1907–1947 covering the periods of all the tables.
2. Crossing and recrossing of some lines that might be due to inconsistencies in the many corrections used for adjusting the age frequencies.
3. The line of the life table of the period 1937–1947 based on the quasi-stable population theory and Model Life-Tables places itself in conformity with the other lines, but it notably deviates from them at the older ages; a fact that will be briefly interpreted in the next section.

The survival rate of males for the life table of the period 1907–1947, which has a life expectancy at birth $\mathring{e}_0$ between 31.8 as maximum and 28.5 years as minimum was plotted, Figure 11, with the two Model Life-Tables of $\mathring{e}_0 = 27.5$ and $\mathring{e}_0 = 32.5$ to envelope the first line. Crossing and recrossing and high deviation at the older ages, of 60 years and over, can again be noted. The same remarks also apply to the females.

The higher survivor rate and expectation of life at the older age-groups can be explained in part at least on age overreporting in these groups. El-Badry says: "it was preferred to use the tail of the 1947 distribution from age 60 upward as the tail of the average distribution in order to utilize what is apparently a systematic improvement in the overestimation of old ages from one census to the next," which overlooks the existence of overestimation in the 1947 census itself.

Overestimation of Old Age-Groups

Census records seem clearly to overestimate the old age-groups in Egypt. The tail of the 1947 census age distribution was analyzed and compared with other countries of higher life expectancy and longer span of life than Egypt and with countries of similar situation to Egypt's population. As age reporting is highly correlated with the literacy status of the population, it seemed desirable to consider some

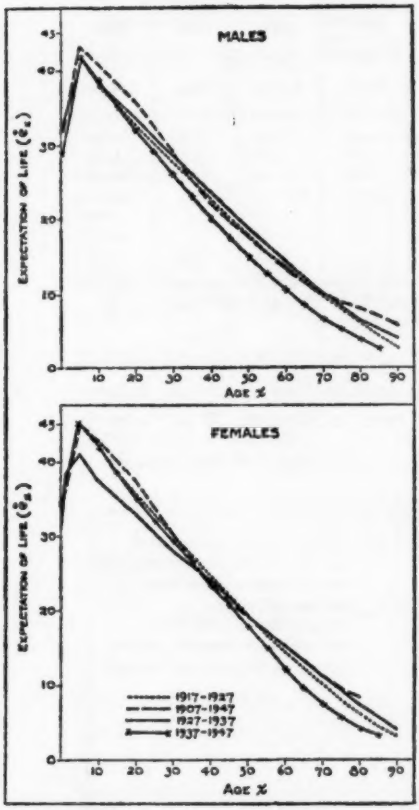


Fig. 10. Expectation of Life ($\mathring{e}_x$) by sex and age in Egypt, as computed in studies for indicated dates.

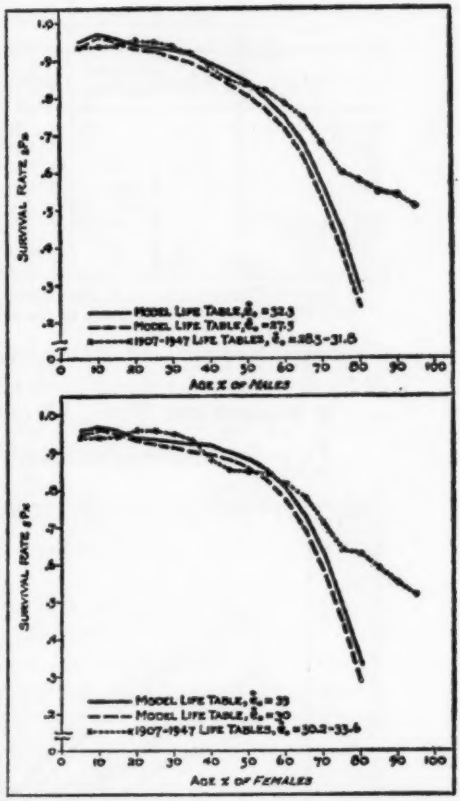


Fig. 11. Survival rates ($_5P_x$) by sex and age in Egypt, as computed in indicated life tables.

countries similar to Egypt in this respect. Moreover two populations that differ in educational and social conditions but in the same country were considered. Censuses dated near to 1947 of the chosen countries were taken.

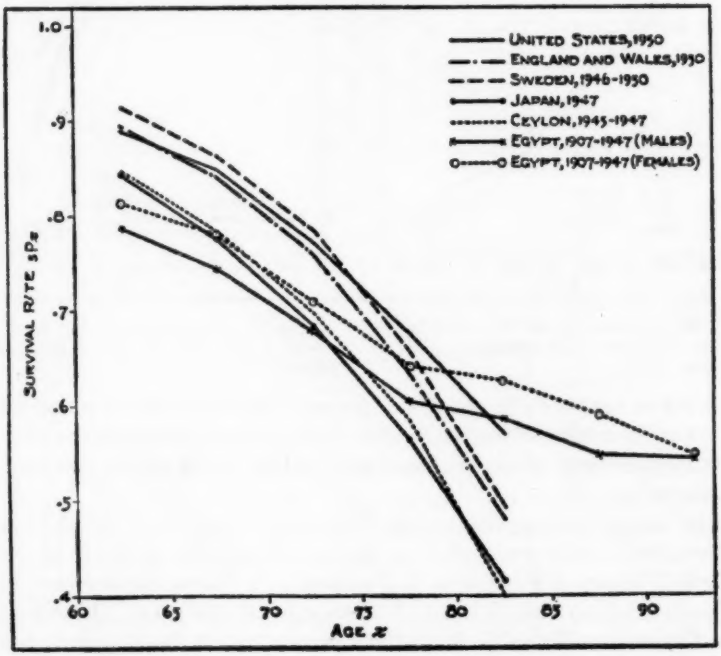
In Egypt's census, those of age 70 years and over were only 2.2 per cent of the total population as compared with 4.8 per cent in the United States, 6.9 per cent in England and Wales, 6.4 per cent in Sweden, 3.2 per cent in Japan, 2.2 per cent in Turkey and 2.1 per cent in Ceylon (see Table 7). With these percentages in mind we find that

Census Year	1950	1951	1950	1945	1947	1945	1946
Age	U.S.	England and Wales	Sweden	Japan	Egypt	Turkey	Ceylon
100+	.06	.01	.01	.01	1.22	1.07	.36
90+	2.04	1.34	1.82	.94	7.21	7.21	4.74
80+	23.45	21.50	23.69	17.67	30.52	30.02	28.34
70+	100	100	100	100	100	100	100
70+ Per Cent Total Pop.	4.83	6.85	6.41	3.17	2.22	2.15	2.05

Table 7. The percentages of population aged 80+, 90+ and 100+ relative to 70+ for some selected countries in the stated censuses.

those aged 90 years and over are 7.2 per cent in Egypt (of the tail 70+) as compared with only 2.0 per cent in United States. 1.3 per cent in England and Wales, 1.8 per cent in Sweden, .9 per cent in

Fig. 12. Survival rates (${}_sP_x$) at ages 60 and over for selected countries. (See Table 8.)



Country	Years	Expectation of Life at Birth $\mathring{e}_0$	Survival Rates $_5p_x$ by Age Groups						
			60–64	65–69	70–74	75–79	80–84	85–89	90–95
United States	1950	68.4	.8907	.8476	.7758	.6774	.5701		
England and Wales	1950	68.9	.8964	.8424	.7593	.6329	.4763		
Sweden	1946–1950	70.3	.9159	.8657	.7834	.6578	.4896		
Japan	1947	52.0	.8461	.7770	.6840	.5634	.4172		
Ceylon	1945–1947	45.8	.8479	.7842	.7013	.5834	.3976		
Egypt (Males)	1907–1947	28.5 31.8	.7880	.7488	.6796	.6043	.5848	.5483	.5441
Egypt (Fem.)	1907–1947	30.2 33.6	.8144	.7795	.7082	.6394	.6265	.5915	.5448

Table 8. Life expectancy $\mathring{e}_0$ and life table survival rate $_5p_x$ to old age groups (both sexes) for some selected countries compared with that in Egypt.

Japan and, as in Egypt, it is 7.2 per cent in Turkey and 4.7 per cent in Ceylon. See Table 7 for further similar comparisons.

As a consequence of this overheaping at old ages in Egypt, its survival rate line, Figure 12, crosses nearly transversally the survival rate lines of the countries whose life expectancies are far higher than Egypt. (See Table 8).

Undoubtedly there is systematic improvement from one census to the next which serves to reduce this overheaping as the literacy and health status of the population are improved. This is the case not only in Egypt but also in other populations. In the United States, the five censuses from 1910 to 1950 show the clear evidence of overheaping in old ages of the non-whites. In the 1910 census, for example, the percentages of 70+ to total population were 2.56 per cent for the whites and only 1.74 per cent for the non-whites, while we see that those of age 90+ in the same census are only 1.57 per cent (of the tail 70+) for the whites and 6.43 per cent for the non-whites. Table 9 shows clearly the overestimation in old ages for the non-whites compared with the white population and how this erroneous population phenomenon is systematically improved from census to census.

Accordingly, the low survival rate (or the expectation of life) for old ages presented by the 1937–1947 life table based on Model-Life Tables and quasi-stable theory seems to be a reasonable estimate for Egypt's population, while the low mortality in the other Egyptian life tables probably has its source in systematic relative overcounts

Census Year	1910		1920		1930		1940		1950	
Age	White	Non-White	White	Non-White	White	Non-White	White	Non-White	White	Non-White
100+	0.04	1.57	0.04	1.50	0.03	1.13	0.00	0.81	0.00	0.66
90+	1.57	6.43	1.56	5.80	1.48	5.24	1.46	4.47	1.78	3.63
80+	20.97	27.97	20.99	26.45	20.29	27.45	21.60	23.58	23.37	23.10
70+	100	100	100	100	100	100	100	100	100	100
70+ Per Cent / Total Pop.	2.56	1.74	2.80	1.87	3.32	1.83	4.12	2.46	5.05	3.03

Table 9. Percentages of white and non-white population of United States aged 80+, 90+ and 100+ relative to 70+ in censuses 1910–1950.

at the older ages. Of course, the higher expectancies at old ages given by the life tables mentioned might express an exceptional character of Egypt's population, but this is very unlikely.

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